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CONTENTS

Pathways for Future Development of Periodontology	279	Irving Glickman
Future Studies on the Experimental Production of Periodontitis in Dogs	284	C. Kenneth Collings David R. Redden
Cartilage and Bone Grafts in Supra-bony Pockets in Dogs	291	E. W. Harder L. B. Thompson
Epidemiology of Diabetes Mellitus: II. A Study of 100 Dental Patients	298	R. C. Sheridan E. Cheraskin F. H. Flynn A. C. Hutto
Editorial	324	
Book Review	325	
Histogenesis of the Gingival Sulcus Epithelium in the Rat	326	G. I. Uohara
Announcements	323, 340, 347, 350	H. R. Schreiber
The Use of Modified Rubber Base Material as a Radiopaque Substance in Periodontal Roentgenography	341	A. Salkand S. J. Ewen
Effect of Calf Bone on Rate of Bone Regeneration in Tibia of Guinea Pigs	348	R. Stallard W. Randall
Index—Volume 30	351	

THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

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Boston, Mass.

Dr. Irving Glickman, Program Chairman for the annual meeting of the American Academy of Periodontology which was held in the Barbizon Plaza Hotel in New York City recently, has long enjoyed an outstanding reputation in the field of periodontology as a clinician, teacher, author and research investigator. He is professor of oral pathology and periodontology at Tufts University School of Dental Medicine, on the staff of the New England Center Hospital, consultant in periodontology at the Forsyth Dental Infirmary for Children, lecturer in oral pathology at Boston University School of Medicine, consultant to the Armed Forces Institute of Pathology, and Director of the Division of Graduate and Postgraduate Studies at Tufts University School of Dental Medicine. Dr. Glickman is a member of many dental organizations, and is a Fellow of the American College of Dentists.

THE JOURNAL OF PERIODONTOLOGY

OCTOBER, NINETEEN HUNDRED FIFTY NINE

Pathways for Future Development of Periodontology*

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DEVELOPMENTS in periodontology in the decade 1949-1959 were numerous and significant. It could appropriately be designated the "Decade of Awareness." In earlier years there had been many reports of the epidemiology of periodontal disease. However it was not until this decade that periodontal disease was recognized as the major menace to the natural dentition of the adult population. In this decade too, educators saw more clearly their responsibility for the teaching of periodontology at the undergraduate and graduate levels.

Realism replaced extremism regarding the role of trauma from occlusion in the etiology of periodontal disease. More research projects were devoted to this problem in the past decade than in the total history of modern periodontology. These studies were in unique accord in corroborating the findings of an earlier generation to the effect that trauma from occlusion, per se, does not initiate gingivitis or periodontal pockets.[1] However, this decade called attention to the fallacy of amplifying this experimental evidence into the broad generalization that excessive functional forces in no way effect the progress and severity of gingivitis and periodontal pocket formation, after they have been started by local irritation. It is recognized that when viewed separately, local irritation from food debris or calculus or food impaction produces microscopic changes in the periodontium which differ in nature and location from those produced by trauma from occlusion. However, when these local factors occur together as is often the case, they are critically interrelated in their combined destructive effects upon the periodontium.

A concept of sensitive interdependence between restorative dentistry and the health of the periodontium crystallized in the past decade. Restorative dentistry was recognized as an important potential cause of periodontal disease. Periodontal disease was established as the major barrier to the longevity of prosthetic restorations. No dentist of this decade could contemplate dental restorations without first restoring periodontal health. Nor could periodontal treatment be considered

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complete without the prosthesis necessary to assure optimum function of the dentition.

Splinting was rejuvenated in the past decade. Multi-unit prosthesis which flourished earlier in the century in central Europe became the vogue in the United States. The development of high-speed tooth cutting added impetus to this trend. Yet to be clarified is the question of whether splinting is actually a therapeutic procedure for tightening loose teeth or simply a mechanical device for holding loose teeth tightly.

Periodontology became concerned with the welfare of the entire dentition as a functioning unit rather than simply with individual teeth. For some, this meant that the welfare of the entire dentition should not be jeopardized by heroic efforts to save questionable teeth. "A prosthetic replacement is no better than the poorest tooth contained within it" and "a healthy pontic is a better neighbor than a questionable tooth" were propounded to check the splinting spree. To others periodontology's concern with the entire dentition as a functioning unit became a mandate for full mouth rehabilitation. Splinting became a diagnostic tool as well as a therapeutic measure. Questionable teeth were splinted as a "test" of their ultimate usefulness. Such teeth were re-evaluated by removing the splint at stated intervals. This was made possible by the use of temporary rather than permanent cementation. Extreme care was urged in the re-examination process to avoid including the tested teeth with the splint as it was removed.

This decade posed the questions "When to splint?" and "What to splint?" and provoked many and sometimes diametric viewpoints in reply.

There were definite developments in the problem of calculus. It came in the form of the introduction of a new and promising experimental approach which shifted the locale of calculus research from the test-tube to the oral cavity.[2] The formation of an adherent muco-polysaccharide and bacteria containing plaque was demonstrated as the initial stage in calculus formation. Calcification was shown to occur in numerous separate foci. The starting time was 3-14 days. It varied in different individuals and from tooth to tooth in the same individual. But by the 14th day there were no plaques without some evidence of some calcification. It was suggested that the carbohydrate protein complexes of the plaque participated in some measure in the calcification mechanism. In in-vitro studies calcification of calculus was inhibited by testicular hyaluronidase.[3]

ENZYMES

Enzymes in the saliva attracted attention. Certain enzymes produced by oral bacteria appeared to be increased in concentration in patients with gingival disease. These include hyaluronidase,[4] betaglucuronidase,[5] chondroitin sulfatase and a substance capable of acting upon collagen preparations.[6] It was suggested that hyaluronidase is of etiologic significance in gingival inflammation,[7] destruction of ground substance and the initiation of periodontal pockets.[8] Investigation of the interrelationship of enzyme activity and periodontal disease was a provocative but not conclusive phase of the decade's activities. This decade continued to probe the bacteriology of periodontal disease and was particularly interested in the etiologic significance of specific bacteria or bacterial groups.

A quarter of a century's acceptance of the "epithelial attachment" concept was challenged,[10] tottered, but did not fall.[11] When the air was cleared the "epithelial attachment" became the "epithelial cuff," attached or adherent.

The role of systemic influences in the etiology of periodontal disease continued to taunt clinicians and researchers. Animal experimentation continued as the principal approach to exploration of possible systemic etiology of periodontal disease.[12] This despite the well understood limitation in the application of findings in animals to dis-

ease as it occurs in humans or even to different species of the same animals.

Hormonal disturbances replaced nutritional deficiency as the major systemic environment induced in laboratory animals. Repeated systemic administrations of cortisone produced osteoporosis of alveolar bone and degeneration of periodontal membrane.[13] The addition of estrogen to cortisone injected animals prevented the osteoporosis and periodontal membrane changes.[14] The periodontium was brought into the orbit of tissues affected by the Stress syndrome.[15,16] Although periodontal changes were not observed in the Alarm Reaction Stage,[17] inhibition of alveolar bone formation was noted in experimental animals after two weeks exposure to cold as a stressor agent.[18] In mice ovariectomized early in adult life, the normal bone formative capacity of the periodontium was observed to be inhibited.

PROTEIN-DEPLETED DIET

Osteoporosis of alveolar bone and reduction in periodontal membrane cell and collagen fiber content ordinarily produced in rats by feeding a protein-depleted diet were prevented by repeated subcutaneous injections of somatotropic hormone.[19] Disorganization and degeneration of the periodontal membrane and impairment in repair induced by occlusal trauma were aggravated by protein depletion.[20] The damage to the periodontium caused by protein depletion alone was repaired by restoration of the normal diet; changes induced by the combination of protein depletion and trauma were not.

Attempts were made in humans to relate systemic findings with the occurrence or severity of periodontal destruction. Despite the fact that it is difficult to consolidate and evaluate the findings of the various investigations because of the lack of uniformity in the criteria whereby the periodontal disturbances are judged, such studies represent important forward steps in the advance of periodontology. A correlation between ovarian dysfunction and the severity of periodontal disease was suggested.[21] Atrophy of the gingiva and alveolar bone was reported in 75% of cyanotic patients with acquired or congenital heart disease.[22]

The importance of protein metabolism in bone behaviour stimulated the undertaking of metabolic balance studies investigating nitrogen, calcium and phosphorous metabolism in relation to alveolar bone changes in patients with and without generalized bone disease.[23] Only five cases were reported but this is a promising start in a necessary approach to periodontal problems. In four patients with generalized metabolic disease the alveolar bone changes and metabolic balance correlated significantly. In a patient with only severe alveolar bone loss, there was a negative phosphorous balance. In another extremely significant study elevated levels of serum glycoprotein in over 40% of a group of patients with periodontosis, stimulated the hypothesis that destruction of the periodontal tissues represented one part of a generalized connective tissue breakdown process.[24]

In this decade, surgery entered the circle of accepted periodontal treatment technics. Snatched from the obscurity where "last resort" treatments abide, the gingivectomy was on its way toward proper appreciation in the early years of the decade.[25] However, what started out as a simple dependable method for eliminating deep periodontal pockets became augmented by a variety of procedures for trimming bone and gingiva[26] in an effort to insure ultimate periodontal health. Perhaps in the next decade the gingivectomy will revert to its less glamorous self. As such it shows promise of fulfilling an important purpose—namely that of providing the general practitioner an effective technic for coping with deep periodontal pockets. Attention was directed to the need for surgical corrective procedures in the region of the mucobuccal fold as adjunctive periodontal treatment measures. Under the connotation of vestibuloplasty there developed a variety

of technics for widening the zone of attached gingiva, deepening the oral vestibule and relocating frena attachments.[27]

There was a marked increase in interest in alveolar bone at the treatment level. Bone trimming and cutting technics of years long gone by were reincarnated. In this decade[28] the technics were modified and modernized by substituting diamond stones for the old bone files and new terminology such as osseous resection, osteotomy and osteoectomy for the less expressive descriptions of the past. As if to balance the bone removing trend, the decade stimulated interest in technics in which bone was added to the periodontium. The use of bone grafts[29,30] of the heterogenous, autogenous, or anorganic types and the use of bone inducers broadened the scope of the treatment of periodontal pockets. "Reattachment" with and without bone grafts was claimed based upon clinical, radiographic and histologic evidence[31,32] but the concept of reattachment did not attain the status of unequivocal acceptance.

The future presents immediate and long range prospects. The nature of calculus may soon be clarified bringing with it nonmechanical preventive and removal agents. Advances on the broad front of geriatrics will bring a better understanding of age changes in the periodontium and their significance in the periodontal problem. More will be learned about saliva in relation to periodontal disease, particularly in regard to age changes in the saliva which foster the increased tendency toward calculus formation with increasing age.

The emphasis in periodontal research will shift from microscopy to biochemistry. Histopathology, long the workhorse of periodontal researchers will be the starting point instead of the goal of investigative programs. Emphasis will shift from morphology to mechanisms and meanings. Instead of repeating experiments which portray the tissue changes induced by occlusal trauma we will advance to explore the metabolic alterations which excessive forces induce in the tissues. There will be a broadside approach to learning more about the physiology of the normal periodontium. A fund of new information will be unearthed about physiologic metabolic activity of the periodontium which has escaped detection by microscopic technics thus far employed.[33] The virology of the oral cavity will be studied bringing a new chapter to the text of dental knowledge. There will be more metabolic studies in humans to learn more about the individual with periodontal disease than the disease itself.

The subject of occlusion will be an important preclinical science in the dental curriculum. The student will be introduced to occlusion as the principal physiologic function of the dentition and not as the blueprint for aligning teeth in artificial dentures. It will be a course in which knowledge of tooth morphology, anatomy of the oral and adjacent structures, muscle and neuromuscular activity will be brought together and integrated so that it has meaning in terms of occlusal function. The day, long overdue, will have arrived when the dental student will know as much about the function of the muscles of mastication as he does about the gastrocnemius of the frog.

Occlusion is the crystallized interrelationship of dental structure and function. It is basic and the same for all branches of dentistry. The restorative dentists, the orthodontist and the periodontist will pool their knowledge regarding occlusion and present it as the common denominator of general dental practice.

New local treatment technics will be tried. New types of instruments will be developed. A place will be sought for new drugs almost as soon as they are produced by the pharmaceutical mills. However, the rationale of local treatment procedures will remain essentially unchanged.

In long range forecast a blurred image of systemic therapy is just perceptible. Anabolic stimulants with specificity of action and without undesirable systemic side

reactions will be developed as physiology unearths new information regarding the basic processes of cellular activity. Without question, these will be explored as adjuncts to local periodontal treatment.

The periodontal record of the past decade is an auspicious one. However, much more progress will be required of the future. In our present situation, periodontal disease is the major menace to the natural dentition of the adult population. The future is challenged to prevent this wasteful tooth loss.

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Further Studies on the Experimental Production of Periodontitis in Dogs

by C. KENNETH COLLINGS, B.S., A.M., D.D.S.,* AND DAVID R. REDDEN, B.S., M.S.,**
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IT HAS been reported that a periodontal lesion resembling periodontitis in the human could be produced in dogs.[1] A technique was described for producing these lesions and roentgenographic and photographic evidence was offered showing the similarity to human periodontal disease.

The present paper is to report further studies upon the reliability of the method; certain variations and controls upon the surgical procedure; and to verify further the uniform success of the procedure in producing the desired results. A method seems to have been found for producing a periodontal lesion similar to the chronic periodontitis and bone loss seen so commonly in advanced cases in the human. Furthermore, this periodontal breakdown can be made to occur very quickly, in 5 to 9 weeks, and with unfailing regularity. This may provide a valuable new source of material, in unlimited amounts which are readily available, for studies of the cause, treatment, and prevention of periodontitis—at least as it appears in an experimental animal.

Much research is being done upon methods of producing periodontal disease. Many approaches are being investigated with interesting results. Ratcliff[2] found evidence of degeneration of collagen fibers and damage to the epithelium in rats which were stressed by immobilization and starvation. Petrovic[3, 4] experimentally produced periodontal disease resembling that found in man, by trauma, infection, and allergic reactions in rats.

Waerhaug[5] studied microscopically the changes produced in the supporting structures of the teeth of monkeys deprived of Vitamin C for periods of from 42 to 237 days. He found histologic signs of disturbances of dentin and bone formation, destruction of collagen fibers, increased osteoclastic resorption and capillary bleeding. He did not believe that the changes seen were like those seen in periodontal disease in man. Schei, Waerhaug, Lovdal and Arno[6] studied bone loss as related to oral hygiene and age in 737 human subjects. They describe a scale or ruler which can be used to measure bone loss on roentgenograms. They found a definite correlation between amount of bone loss and age and condition of oral hygiene.

Person[7] produced gingival lesions in guinea pigs by injecting homogenized gingival crevice scrapings from human mouths. He found that the severity of these lesions could be increased when a major energy-supplying metabolic process was blocked or when animals were starved. Goldman[8] demonstrated a degeneration of the attachment apparatus, including bone loss and osteoporosis, in rats and spider monkeys following dietary protein deprivation. The effects seemed to be much more evident in young animals than in old ones. Fransden, Becks, Nelson and Evans[9] demonstrated that significant periodontal defects in young rats could be produced by feeding protein deficient diets.

Glickman and Weiss[10] cemented onlays on the anterior teeth of dogs in order to study the role of trauma from occlusion in initiation of periodontal pocket formation. They found that trauma from occlusion did not initiate the formation of periodontal pockets in dogs. The onlays were made and mounted so that the forces were parallel to the long axes of the teeth. Wentz, Jarabak, and Orban[11] devised a mechanism which would produce traumatogenic displacement laterally in opposite directions. By means of springs, they were able to pull a tooth back from its position when displaced laterally by another traumatic force. This movement they called a "jiggling" movement. They describe the typical results of traumatogenic force upon the periodontium in early stages (up to 3 weeks) but after as much as 6 months, all damage was resolved except for widened periodontal ligament which they interpret as a sign of tissue adaptation. They leave the question unanswered as to what might have happened had some local irritant, such as calculus, been present.

Beube[12] prepared localized pockets on canine teeth of dogs by reflecting the gingiva and removing the buccal plate surgically in 6 millimeter squares. He found progressive regeneration of bone and supporting tissues over a period up to 2 years. No

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evidence was offered that any progressive bone loss occurred following his surgical procedures. Yuktanandana[12] produced intrabony pockets in dogs by using 701 or 702 burs to cut out bone and supporting tissue adjoining selected teeth. The pockets were packed with acrylic, tin foil, or surgical cement for one week to prevent healing. At this time, autogenous bone grafts were made and healing was observed by microscopic studies. He does not mention any evidence of further loss of bone following his surgical method.

Schaffer[14] created bone loss in a dog and a Rhesus monkey by turning a flap of soft tissue and removing bone with a bur between two adjoining teeth. He later implanted shavings from the root surfaces of teeth into the pocket and observed the healing process. No mention is made of further bone loss. Schaffer[15] also produced pockets in Rhesus monkeys by a similar method and studied healing subsequent to cartilage transplants. Again, no mention is made of bone loss following surgical procedure.

Boyle[16] described a dog which was found to have osteoporosis and bone resorption of the jaws but no evidence of such lesions elsewhere. Carranza and Carranza[17] list those conditions found in the human alveolar bone which should be treated by surgical removal of the offending bone. They express the opinion that it is preferable not to disturb alveolar bone. Baer[18] has reported a genetic strain of white mice which consistently shows periodontal disease. The rice rat is also reported to suffer from periodontal disease.[19]

The preceding survey of the literature reveals some interesting trends in thought of the researchers into periodontal disease. It seems that most efforts to produce periodontal disease take the form of inducing metabolic disturbances in the animals. Stress, diet, metabolism, and vitamin imbalance have all been utilized by the above investigators as systemic influences.

Others use local traumatizing effects. These investigators, in most cases, are interested in studying the local effects of excessive loads upon the supporting structures of the teeth. And still others produce the periodontal pockets by surgical removal of tissue for the purpose of studying the regeneration and healing of the area.

None of the above procedures causes a widespread periodontal bone loss and inflammation such as occurs following the technique described in this report.

METHODS

At the time of the publication of the earlier paper on this subject, seven dogs had been used and observed over periods of from four months up to seven years. No effort had been made to determine the time factors in the development of the periodontitis following surgery. In an effort to determine the rapidity with which the condition developed, a pilot series of seven dogs was treated as described in the preliminary report.[1] Each dogs was anesthetized with Nembutal and the free and attached gingiva was reflected buccally and lingually from around the premolars and the first molars on both the right and left sides of the mandible. A narrow sliver of bone, between one and two millimeters in width, was removed from the crest of the alveolar bone on both the buccal and lingual of the exposed areas, using a sharp surgical chisel and a mallet. A small bone bur was used in a handpiece, at a rather slow rate of rotation, to smooth the floor of the shelf in the bone which had been created by the chisel. This bur was applied quickly and intermittently in an effort to minimize any frictional or heat effects.

The soft tissue was now replaced and sutured between the teeth. The dogs were fed a gruel, composed of canned dog food stirred into whole milk, so thin that the animal had to lap up the food. This avoided any chewing which might have torn out the sutures. The sutures were removed after about two weeks and the dogs returned to a diet consisting of table scraps and Purina Dog Chow.

Roentgenograms of the areas involved were made before surgery, immediately following surgery, and at various intervals later, as necessitated by the nature of the experiment. Also, Kodachrome photographs were made at intervals. This entire procedure was basic to all experiments. Variations were introduced later as will be explained.

These seven dogs were sacrificed, one

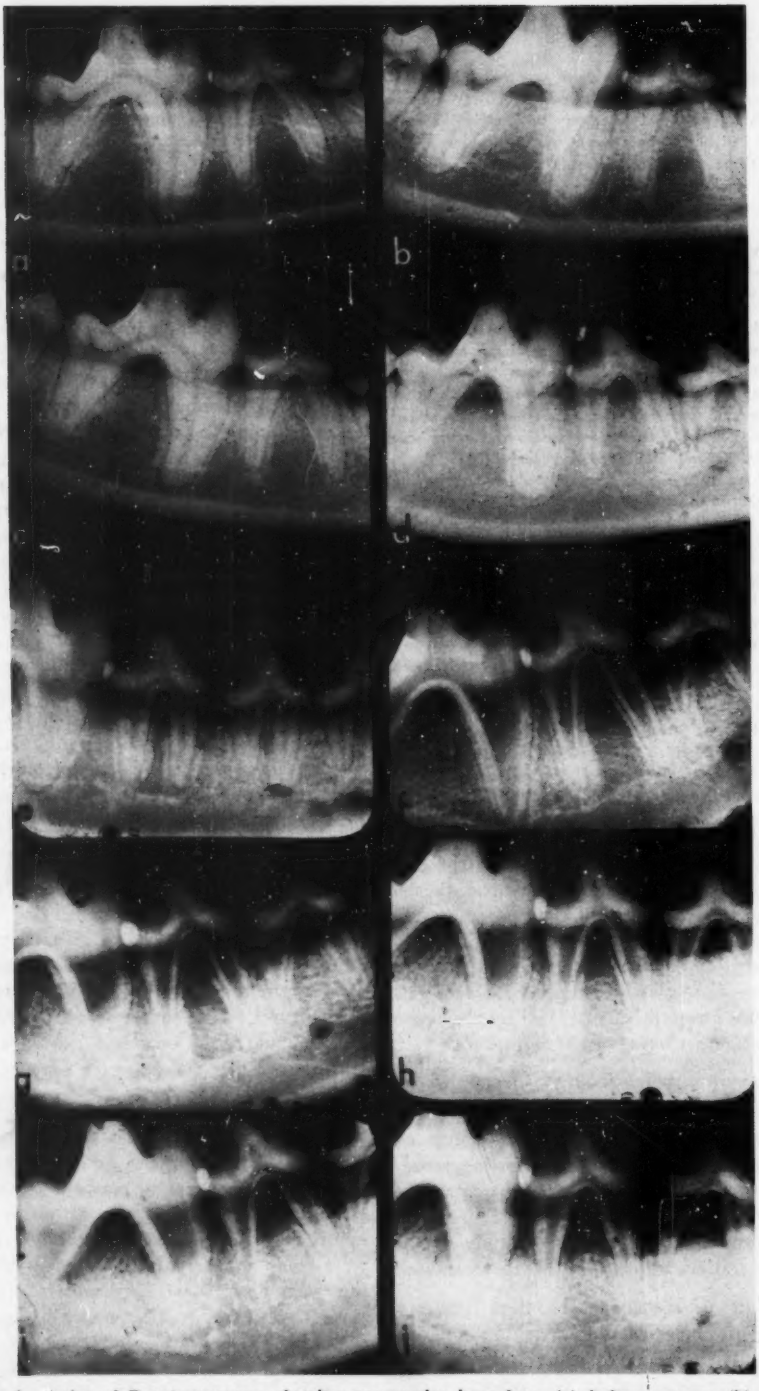

Fig. 1. Series of Roentgenograms showing progressive bone loss. (a) before surgery (b) after surgery (c) 7 days (d) 14 days (e) 24 days (f) 28 days (g) 35 days (h) 42 days (i) 63 days (j) 16 weeks.

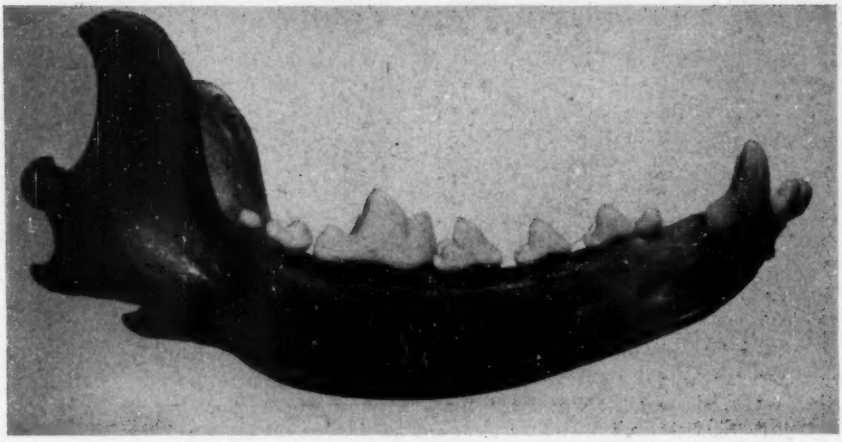

Fig. 2. Mandible two and one-half weeks after surgery.

each time, at intervals of 1, 2, 3, 4, 5, 6 and 9 weeks. Roentgenograms and photographs were made just preceding death. The mandibles, with the investing soft tissues, were excised and were preserved, in small sections, in standard fixing solutions for use for microscopic study. These observations will be reported in a subsequent paper.

After examining the above roentgenograms, another series of nine dogs was operated as above, except as follows:

1. Surgery was done on the right side only, the left side being left untouched as an individual control.

2. Roentgenograms and photographs were made on these animals beginning on the fourth week and repeated at five, six, and nine weeks.

3. All these dogs were permitted to live and will be used in subsequent studies.

In the experimental method described, several separate steps were necessary, any of which might have been the one causing the bone loss. In an effort to isolate each step, the following five variations of the procedure were carried out as additional controls:

1. A flap, as described, was turned and the bone was left exposed to the air a length of time corresponding to the time needed for a complete operation. The flap was then sutured back into place.

2. A flap was turned and a blunt instrument, instead of a chisel, was tapped against the surface of the marginal bone, using the same mallet with about the same force as necessary to chisel. The flap was then sutured back into place.

3. A flap was turned and a burnishing bur was applied to the surface of the marginal bone, rotating at about the same speed as the bone bur had been used. The friction and heat of this were undoubtedly greater than that obtained with the bone bur. The flap was sutured back into place.

4. An incision was made, bucally and lingually, just apical to the muco-gingival line of attachment and the flap of soft tissue was turned upward so that the investing soft tissue around the necks of the teeth was not incised. Following this, one method was to chisel the bone as before and then replace the soft tissue. Another method was to turn the flap upward, freeing it from the bone, and then to immediately replace it, without anything being done to the bone.

5. In all cases of alveolar bone removal

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Fig. 3. Mandible 27 weeks after surgery.

a sharp-edged shelf had been produced on the crestal bone. To eliminate this as a possible cause of bone resorption, a flap was turned, bone removed and the sharp edge rounded to a good physiological contour using a coarse diamond stone. The soft tissue was then sutured into place.

A total of twenty-seven mongrel dogs was used in the above series of experiments. We feel that this is a significant number, especially when added to the seven dogs reported in our earlier paper.

RESULTS

The first series of seven dogs, in which the development of the periodontitis was studied, showed a remarkably rapid and consistent periodontal destruction. Until microscopic studies can be made and reported later, the evidence submitted in this paper will be largely roentgenographic. Fig. 1 shows a series of roentgenograms of the mandibles of dogs made at the intervals indicated. Fig. 1a shows the area on the mandible before any surgery was performed. Fig. 1b shows the appearance of the bone in the same dog immediately following surgery. A distinct line can be seen slightly below the crest of the bone. This represents the edge of the shelf of bone created buccally and lingually by the chisel and bur. It is interesting to note that, in all of our roentgenograms, a well-defined shelf was visible in all the pictures made immediately following surgery. Fig. 1c shows the same animal seven days later at the time it was sacrificed. The shelf of bone is still plainly visible. Fig. 1d shows the appearance of the next dog in the series at the time of sacrifice fourteen days after surgery. The shelf of bone is still visible, although it may appear to be somewhat less distinct. Fig. 1e shows the third animal in the series after twenty-four days. The sharp line of the shelf has been replaced by a zone of radiolucency. This zone of radiolucency appears to be comparable to that seen so frequently in roentgenograms of typical active cases of human periodontitis. The radiolucency is greatest at the crest of the bone, but lamina dura and trabeculae are still faintly

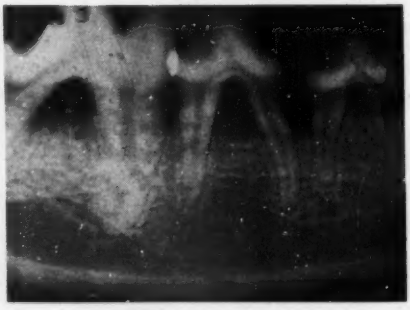

Fig. 4. Roentgenogram of mandible in figure 3.

Fig. 5. Mandible showing infrabony pockets.

visible, particularly in the interradicular areas. It has the appearance of bone which is being rapidly destroyed. Fig. 1f shows the fourth animal in the series at twenty-eight days. The area of radiolucency at the crest of the bone seems to be considerably wider and to extend deeper into the bone. The premolar tooth on the extreme right of the roentgenogram appears to have a bifurcation involvement which penetrates entirely through the alveolar crest.

Fig. 1g is of the same dog at thirty-five days. A margin of rarefaction shows which is comparable to that at twenty-eight days. Fig. 1h shows the same dog after forty-two days. This roentgenogram shows the variations that appear in series of pictures taken at different times. Fig. 1h seems to show a regression in the severity of the periodontitis from Fig. 1g. Actually, this must be a variation in roentgenographic technic, since later exposures reveal even greater bone loss. Efforts were made to standardize our technic, but variations may possibly appear. The zone of rarefaction in Fig. 1h does not appear to be distributed as deeply into the bone as in Fig. 1g, but, rather, there is a sharp line of demarcation between the affected and unaffected bone in the region of the most mesial premolar shown. It would seem to indicate that the bone destruction had been slowed or even arrested at this level around certain teeth.

Fig. 1i is the same dog after nine weeks (sixty-three days). It can be seen that the condition has now advanced considerably beyond that of six weeks (forty-two days) shown in Fig. 1h. The line of demarcation is still abrupt and the level of bone destroyed is approximately the same. There is no bone visible in the bifurcations of the premolar teeth. Fig. 1j shows the same dog after sixteen weeks. It appears that the bone loss has progressed further but at a slower rate than during the first six weeks following the surgery.

In the second series of nine dogs, in which surgery was performed only on the right side of the mandible, the left side being left as a control, the same sequence of events as in the first series appeared. The operated side, at successive weekly examinations, showed the same pattern of destruction, as described, while no bone loss occurred on the unoperated side at any time.

In the series of dogs used in the experiments to determine if any one part of the surgical procedure was the chief cause of the observed phenomenon, it seemed that the removal of the thin slivers of bone was the determining factor. In no case was any roentgenographic evidence of bone loss visible unless the slivers of bone were removed. Trauma from a blunt instrument, friction from a burnishing bur, exposure to air or the incision in the free margin caused no detectable bone loss by the method of observation used in these experiments. Turning the flap from the muco-buccal fold coronally produced no bone loss. Also, smoothing the sharp margin of the shelf of bone did not delay or prevent the radiographic evidence of periodontal disease. From these control experiments, we feel justified in concluding that the removal of bone is the critical factor.

Fig. 2 shows the mandible from a dog sacrificed two and one-half weeks after experimental surgery. This mandible demonstrates an appearance of bone comparable to that shown in the roentgenograms of the dogs in Figs. 1d and 1e. Bone is still present in the bifurcations and interproximals, but cortical bone has been lost along the line where the sliver was removed surgically.

Fig. 3 shows the mandible from a dog

sacrificed at twenty-seven weeks. Cortical bone loss has progressed considerably further apically; the trabeculae of the underlying cancellous bone are visible and the bifurcations are perforated. Fig. 4 is a roentgenograph of this mandible taken in the living dog immediately preceding sacrifice.

Fig. 5 shows the mandible of a dog sacrificed four years after the experimental surgery had been performed. This is included in this report to show the occurrence of infrabony pockets in dogs in which this condition is allowed to develop over a long period. The same infrabony pocket-type of bone loss was observed in two additional mandibles from dogs sacrificed at five years and at six years respectively. The present series of mandibles does not show at what age infrabony pockets start to develop.

SUMMARY

1. A method of experimentally producing periodontitis in dogs is further investigated.

2. The destruction of supporting structures is slow during the first two weeks following surgery; is very rapid during the next three to six weeks, then progresses very slowly during many months following.

3. The critical part of the surgical procedure is the removal of the slivers from the crest of the bone buccally and lingually to the posterior mandibular teeth.

4. Histologic sections have been prepared and a report on the microscopic changes will be made.

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Cartilage and Bone Grafts in Supra-bony Pockets in Dogs

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THE ultimate objective of periodontal therapy is the replacement of attachment apparatus which has been lost through periodontal disease. New impetus to this objective has been achieved through works of Ramfjord,[1] Linghorne and O'Connell,[2] Schaffer,[3,4,5] Cross,[6] Melcher,[7] and Prichard.[8] While previous emphasis has been placed on seeking a new attachment within the confines of the infra-bony pocket, this investigation was an attempt to gain new attachment at a higher level than the alveolar bone present.

PROCEDURES

Modified flaps were made on the palatal and labial surfaces of the maxillary upper canine teeth of dogs. With the flaps retracted, approximately five millimeters of alveolar bone were removed around the entire circumference of the canine tooth. Either a surgical pack or adhesive tin foil was placed around the exposed cementum of the tooth down to the new alveolar bone level. The flaps were sutured in position. After two weeks the sutures and surgical pack or tin foil were removed. Pockets of various depth, with characteristics similar to those found in periodontitis, were present.

After intervals of several months, modified flaps were again made on the palatal and labial surfaces of the previously operated canines. The exposed cemental surfaces were thoroughly scaled and planed to the alveolar bone level. A more vigorous planing was performed at the alveolar bone level, so a definite groove would be present. The epithelial lining on the pocket side of the modified flaps was removed through curettage. In three dogs, homogenous costal cartilage chips stored in 70% alcohol were packed against the planed cemental surface from the alveolar bone level to the height of the modified flap. In four other dogs, autogenous or homogenous alveolar bone chips preserved in 1:1000 aqueous merthiolate were similarly packed against the planed cemental surface from the alveolar bone level to the height of the modified flap. The packing of the cartilage chips or bone chips was done on either the labial or palatal surface of each canine so that the opposite side would serve as the control side. The flaps were sutured in position. Adhesive tin foil was placed around the tooth to protect the sutures. A fast setting acrylic stent was placed over the tin foil to further protect the operated areas. The acrylic stents, tin foil, and sutures were removed in two weeks.

The animals were sacrificed at various intervals. Each canine tooth operated was removed in block section complete with soft tissue and maxillary bone. The sections were fixed in 10% formalin. They were decalcified in a solution of equal amounts of 20% sodium citrate and 50% formic acid. Paraffin sectioning was done at 10 micra. Hematoxylin-eosin, Mallory, and silver staining techniques were used.

HISTOLOGIC RESULTS

Fig. 1. Normal dog periodontium. This is a section illustrating the histological features of the periodontium of a maxillary canine of the dog, and in most respects resembles the human periodontium. Fig. 1A is a section under higher magnification of the palatal surface showing epithelial attachment, periodontal ligament fibers, cementum, cementoid, cementoblasts, alveolar bone, osteoid and osteoblasts. Fig. 1B is a similar section on the labial surface.

Fig. 2. Surgically created pockets. Fig. 2 is a five millimeter pocket on the palatal

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Fig. 1

surface. The crevicular epitheluim has proliferated down along the pocket surface. An intense chronic inflammatory reaction is concentrated heavily along the pocket epithelium. Fig. 2A is the pocket on the labial surface.

Fig. 3. Homogenous cartilage graft in labial pocket, five weeks duration. Fig. 3 is the palatal pocket and retains its original depth to the deepest part of the root curettage. Fig. 3A is the labial surface. New periodontal fibers can be seen in the newly formed cementoid. These fibers appear to be running from the deepest area of root curettage coronally and parallel to the root surface. The cartilage grafts can be seen embedded in periodontal fibers, and one piece of cartilage in the field is surrounded by osteoid material.

Fig. 4. Homogenous cartilage graft in palatal pocket, seven weeks duration. Fig. 4 is the palatal area and illustrates a new attachment of epithelium to the planed root surface. Fig. 4A is the labial pocket and no new attachment has been formed.

Fig. 5. Homogenous cartilage graft in labial pocket, twelve weeks duration. Fig. 5 is the palatal surface. A small piece of cartilage graft has been forced from the labial to the palatal surface and some new attachment apparatus has been stimulated

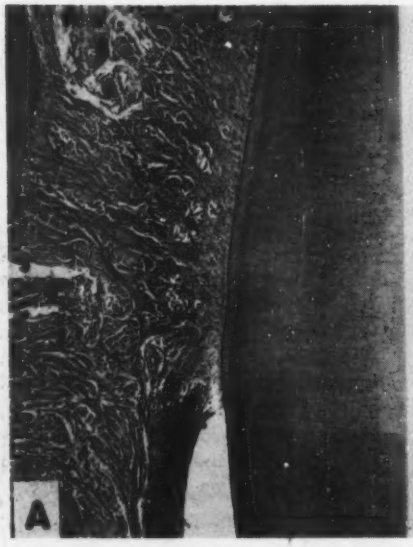

Fig. 1A

Fig. 1B

Fig. 2

Fig. 2A

to form. Fig. 5A shows a large area of new attachment, epithelial as well as connective tissue. Extensive cementoid material is present in the area of root curettage. The cartilage grafts are surrounded by large areas of new bone formation.

Fig. 3

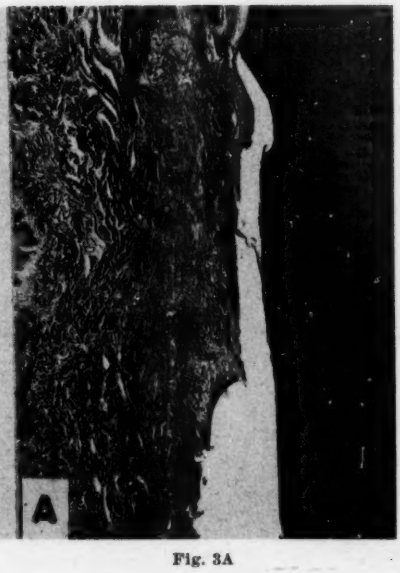

Fig. 3A

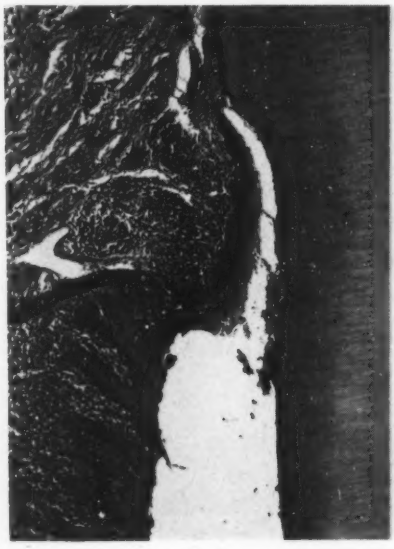

Fig. 4

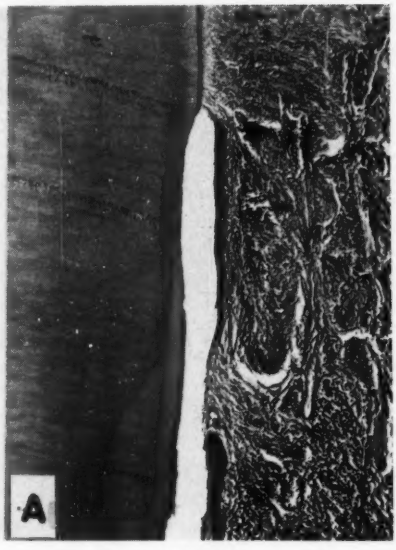

Fig. 4A

Fig. 6. Homogenous bone graft in palatal pocket, two weeks duration. Fig. 6 is the palatal or grafted surface. This section exhibits an acute inflammatory reaction. The pocket epithelium has proliferated upward and surrounds the grafted bone chips which are being expelled into the pocket space. A small bone chip that has not been

Fig. 5

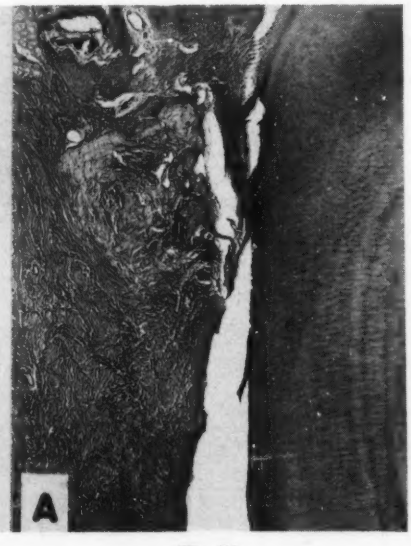

Fig. 5A

Fig. 6

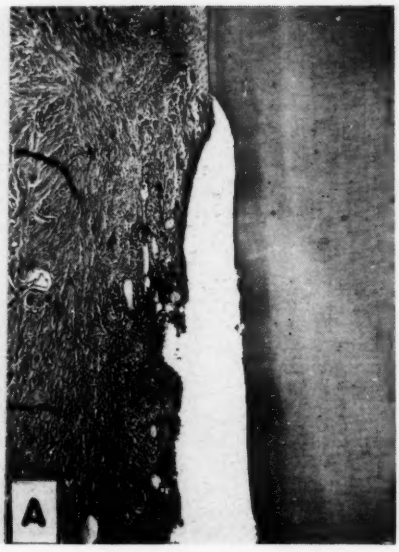

Fig. 6A

expelled is surrounded by osteoclasts and is undergoing resorption. The acute inflammatory reaction present precludes any attempt to form a new attachment. Fig. 6A is the labial pocket and is present to its original depth.

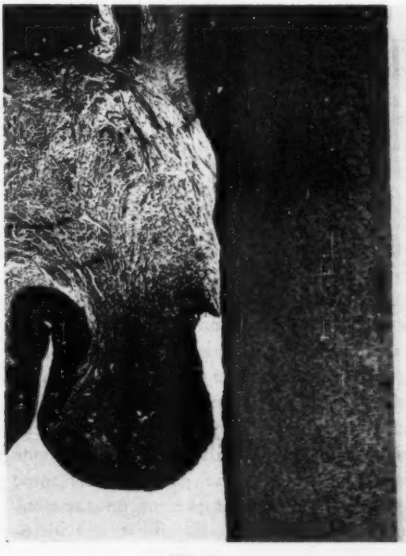

Fig. 7

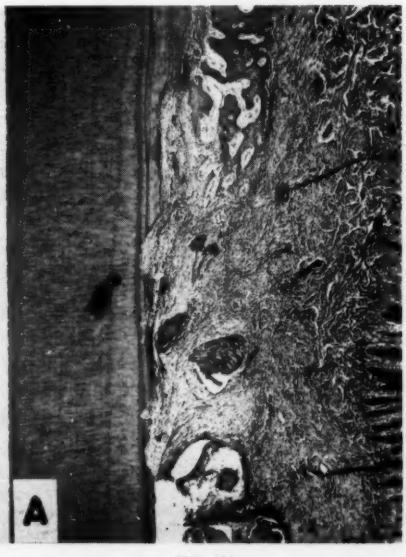

Fig. 7A

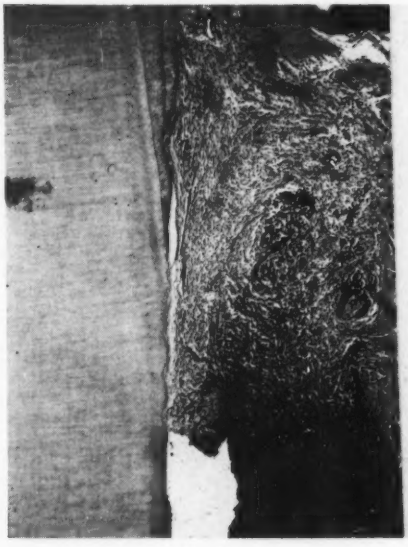

Fig. 8

Fig. 8A

Fig. 7. Autogenous bone graft in labial pocket, four weeks duration. Fig. 7 is the palatal or control side, and contrary to expectations, a small area of new attachment has been formed. Fig. 7A is the labial or grafted side and this section shows considerable area of new attachment. While coronally some bone chips are being expelled, at a higher level the bone chips are circumscribed by osteoid material. At a still higher level, new alveolar bone has been laid down. New periodontal fibers can be seen for considerable distance running parallel and embedded into newly formed cementoid. A long layer of cementoblasts can be seen lining the cementoid.

Fig. 8. Autogenous bone graft in palatal pocket, six weeks duration. Considerable new attachment apparatus has been formed on the grafted side. Fig. 8A is the labial or control side. At the base of the curetted area some cementoid has been laid down and new periodontal fibers are present.

Fig. 9. Homogenous labial bone graft, eight weeks duration. Fig. 9 is the palatal surface and shows no new attachment. Fig. 9A shows new attachment apparatus formed on grafted side. The bone graft is completely surrounded by osteoid and osteoblasts. New attachment of connective tissue and epithelium is present.

DISCUSSION

It is apparent that a new attachment apparatus was stimulated to form with cartilage or bone grafts in six of the seven cases of supra-bony pockets in dogs. A maximum of three millimeters of new attachment apparatus was attained as ascertained from direct measurements on the slides. One received the impression from a study of the histologic sections that the bone grafts, autogenous or homogenous, have a greater stimulatory effect on new attachment apparatus than cartilage grafts. It is possible, however, that this result might be due to improved grafting technique, since the cartilage grafts were done previous to the bone grafts. That some new attachment was present on the control or non-grafted side, illustrates histologically that some new attachment can

Fig. 9

Fig. 9A

be achieved through subgingival curettage procedures alone in supra-bony pockets. This result corroborates clinical impressions of similar procedures in human patients.

It would seem that similar grafts of cartilage or bone could be used successfully in human subjects, in selected cases, to rebuild attachment apparatus lost through periodontal disease. It should be possible to obtain and preserve human nasal cartilage from our ENT associates. Similarly, alveolar bone particles are available from oral surgeons as they perform alveolectomies and removal of exostoses.

SUMMARY

Periodontal pockets were surgically created about maxillary canine teeth in dogs. Subgingival curettage was performed in these areas. Homogenous costal cartilage and autogenous or homogenous alveolar bone grafts were placed in either the palatal or labial supra-bony pockets. After various intervals, the dogs were sacrificed and the sections studied histologically. New attachment apparatus up to three millimeters was attained. Similar grafting techniques should be applicable in human periodontal disease.

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Epidemiology of Diabetes Mellitus: II. A Study of 100 Dental Patients*

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IN an earlier report,[1] the available literature for the past two decades regarding the oral manifestations of diabetes mellitus and the present laboratory screening criteria for the detection of diabetes mellitus were reviewed briefly.

This report is a result of a study of 100 routine dental patients who visited the University of Alabama School of Dentistry for oral examination and care. These individuals were studied carefully for the presence of extra- and intraoral symptoms, intraoral signs, and oral roentgenographic findings by a dental examiner (R.C.S.). The screening technique used for the detection of diabetes mellitus consisted of the 3-hour *true* glucose tolerance test.

By these means, an attempt will be made to answer the following four questions:

1. What is the prevalence of diabetes mellitus in a *dental* population? In other words, how often does an *unrecognized* diabetic patient appear in the dental office?

2. What is the prevalence of *oral* findings of diabetes mellitus in a *dental* population? In other words, what symptoms and signs will the *unrecognized* diabetic patient appear with when he presents himself for oral examination and care?

3. Among nondiabetic patients, is there any correlation between blood glucose levels and *oral* symptoms and signs? In other words, can one observe relationships in the oral cavity to justify the term *prediabetes mellitus*?

4. What is the prevalence of the antithesis of diabetes mellitus? In other words, what are the *oral* findings in patients with *flat* glucose tolerance patterns?

METHOD OF INVESTIGATION

A series of 100 presumably healthy patients were studied. These individuals all reported to the Screening Section of the University of Alabama School of Dentistry for examination and care. The patients who were referred to the Section on Oral Medicine for participation in this study were *not* selected at random according to the usual sampling method.[2] True random sampling was not possible because it was necessary to obtain patients who could devote the three-hour period for the glucose

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tolerance test, the additional clinical examination and the extra roentgenographic study. All of the individuals in this investigation were analyzed for: (1) *general characteristics*, (2) *history*, (3) *clinical (oral) examination*, (4) *intraoral roentgenograms*, and (5) *laboratory procedures* in the form of a three-hour true glucose tolerance test.

GENERAL CHARACTERISTICS

General information was obtained from each patient including: (1) *age*, (2) *sex*, (3) *race*, (4) *whether the patient was aware of his diabetic status*, and (5) *reason for admission* to the dental clinic (Figure 1).

Age: The general statement has been made that diabetes mellitus may appear at any age. However, the curve of incidence is at its maximum between the ages of 40 and 60.[3] Accordingly, age was recorded as shown in Fig. 1.

Sex: It has been generally recognized that, between the ages of approximately 40 to 70 years, diabetes mellitus is significantly more prevalent in women than in men with the ratio being approximately 3 to 2.[4] The sex of the individual was recorded as shown in Fig. 1.

Diabetic Status: Each patient was questioned as to whether he or she had ever been told that they were diabetic (Fig. 1). If diabetic, questioning was pursued to determine the precise status in terms of diet and hypoglycemic therapy.

Reason for Admission: As far as can be determined, no attempt has ever been made to establish the type of *oral* complaints which bring *diabetic* patients into the *dental* office. One *medical* study of 355 diabetic patients[5] has shown that xerostomia is the most common complaint and that it is reported by 3 out of every 4 diabetic individuals. Fig. 1 shows the scoring system utilized for recording the reason for admission based on a 10-point scale.

HISTORY

It should be clear from the review of the literature in connection with oral findings[1] that symptoms, in association with diabetes mellitus, have been inadequately investigated. In the main, the observations in the literature are quite gross and seldom are specific figures cited. It is true that *symptoms* are difficult to evaluate. Accordingly, in this study very gross scoring systems were used for most of the evaluations. However, in some instances (e.g. gingival bleeding) more elaborate scoring was used (Fig. 1).

In the main, those symptoms were investigated which could be elicited by a dental examiner. These may be divided into *two* categories: (1) *extraoral symptoms*, and (2) *intraoral symptoms*.

Extraoral Symptoms: The items which were studied include: (1) *family history of diabetes mellitus*, (2) *boils*, (3) *appetite*, (4) *fluid intake*, (5) *fluid excretion*, and (6) *weight change*.

Family History of Diabetes Mellitus: The genetic predictability of diabetes mellitus has been charted.[6] The evidence indicates that, if both parents are diabetic, there is a 100 percent chance that the offspring will also be diabetic. If, on the other hand, one parent and the grandparent on the other side is diabetic, then the percentage decreases to 61. If but one first cousin is suffering with diabetes mellitus, then the likelihood of the patient also having diabetes decreases to 9 percent. All of these possibilities have been studied by these investigators.[6] Possible diabetes mellitus in the family was recorded as shown in Figure 1.

Boils: Recurrent furunculosis or boils have been recognized as a frequent diabetic finding.[7] The presence of this condition was recorded (Fig. 1).

Appetite: The classical description of diabetes mellitus includes the statement that there is frequently an increase in appetite.[8] The possibility of an increase or decrease in appetite was charted (Fig. 1).

Fluid Intake: Frequent reference is made to polydipsia as part of the diabetic syndrome.[8] An increase or decrease in fluid intake was recorded (Fig. 1).

Fluid Excretion: Polyuria is regarded as one of the common diabetic findings.[9] Accordingly, a record was kept of possible increase or decrease in urinary excretion (Fig. 1).

Weight Change: The classical descriptions of diabetes mellitus include mention of the fact that loss of weight is a rather characteristic accompaniment of diabetes mellitus.[8] For this reason, a record was kept as to whether the patient lost or gained weight within the preceding six month period (Fig. 1).

Intraoral Symptoms: Mention has been made in the preceding report[1] that the most common subjective complaints reported in the literature are: (1) *dry mouth*, (2) *burning mouth*, (3) *tender gingiva*, (4) *pain with percussion of the teeth*, and (5) *dry sockets*. Accordingly, these same items have been included in the present study.

Dry Mouth: It is extremely difficult to evaluate xerostomia, and this may well explain the paucity of quantitative data in the literature.[10] In this study dry mouth was recorded in four categories (Fig. 1).

Burning Mouth: This symptom has also been associated with diabetes mellitus.[10] Since it is extremely difficult to evaluate this complaint, burning mouth was recorded in three categories (Fig. 1).

Tender Gingiva: Frequent mention has been made of this complaint in the literature but no figures are cited.[11-17] Because of the difficulty in evaluating this

FIGURE 1

Name_____

Chart number_____

Group_____

GENERAL CHARACTERISTICS

Age ☐ ☐

Sex ☐
1—female
2—male

Race ☐
1—negro
2—Caucasian

Diabetic ☐
1—no
2—yes

Reason for admission ☐
0—routine check
1—loose teeth
2—sore gums
3—sore teeth
4—sore tongue
5—sore cheek
6—diabetic survey
7—periodontia
8—teeth filled
9—teeth extracted

Patient's number ☐ ☐ ☐

Photographs ☐

Roentgenograms ☐

EXTRAORAL SYMPTOMS

Family diabetes ☐
0—absence
1—father
2—mother
3—brother
4—sister
5—aunt
6—uncle
7—combination of two
8—combination of three
9—all

Boils ☐
0—absent
1—occasional
2—constantly
3—unknown

Appetite ☐
0—same
1—increased in last six months
2—decreased in last six months
3—unknown

Fluid intake ☐
0—same
1—increased last six months
2—decreased last six months
3—unknown

Fluid excretion ☐
0—normal
1—increased last six months
2—decreased last six months

Weight change ☐
0—same
1—gained last six months
2—lost last six months

INTRAORAL SYMPTOMS

Dry mouth ☐
0—absent dry mouth
1—occasional
2—constantly
3—unknown

Burning mouth ☐
0—absent
1—occasional
2—unknown

Tender gingiva ☐
0—absent
1—occasional
2—constantly
3—unknown

Pain with tooth percussion ☐
0—absent
1—slight sensitivity
2—severe pain
3—unknown
4—edentulous

Dry sockets ☐
0—absent
1—present
2—unknown

INTRAORAL SIGNS

Dryness of lips ☐
0—no dryness
1—mild dryness
2—severe dryness
3—questionable dryness

Gingival stippling ☐
0—normal
1—slight loss of stippling
2—moderate loss of stippling
3—severe loss of stippling

Gingival bleeding ☐
0—no gingival bleeding
1—bleeding with toothbrushing
2—bleeding upon biting into apple, pear, etc.
3—bleeding upon biting into a sandwich, roll, etc.
4—spontaneous bleeding

Gingival hue ☐
0—coral pink
1—red
2—blue to purple

Pocket formation ☐
0—normal gingival sulcus
1—absolute pockets 1-3 mm. deep
2—absolute pockets 3 or more mm. deep
3—relative pockets 1-3 mm. deep
4—relative pockets 3 or more mm. deep
5—absolute and relative pockets 1-3 mm. deep
6—absolute and relative pockets 3 or more mm. deep
7—edentulous

P. M. A. Index ☐
0—no clinical evidence of inflammation
1—detectable hyperemia in the papillae, marginal or attached mucosa
2—loss of stippling, redness, swelling or bleeding upon pressure
3—the patient also complains of symptoms such as bleeding, sensitivity, itching sensation, or tenderness
4—presence of severe hyperemia or obvious swelling, spontaneous hemorrhage or bleeding following the slightest contact with food or toothbrush
5—edentulous

Smooth tongue ☐
0—normal
1—slight loss of papillae at tip
2—moderate loss
3—generalized smoothness

Geographic tongue ☐
0—no denudation
1—mild patchy areas
2—severe patchy areas

Tongue color ☐
0—pink
1—mild red
2—bright red

Tongue size ☐
0—no marginal indentations
1—slight marginal indentations
2—marked marginal indentations

Tongue fissures ☐
0—none
1—slight
2—severe

Tooth mobility ☐
0—no tooth mobility
1—less than 1 mm.
2—more than 1 mm.

Number of teeth ☐ ☐

Calculus ☐
0—absence
1—slight supragingival
2—moderate supragingival
3—slight subgingival
4—moderate subgingival
5—slight supra- and subgingival
6—moderate supra- and subgingival
7—marked supra- and subgingival

ROENTGENOGRAPHIC EXAMINATION

Alveolar bone loss ☐
0—normal septal crest
1—minimal alveolar bone loss
2—minimal to moderate alveolar bone loss
3—one-half the alveolar support missing
4—moderate to severe alveolar bone loss
5—no alveolar support

Widening of the periodontal membrane ☐
0—no marginal widening
1—minimal marginal widening
2—severe marginal widening

LABORATORY DATA

Fasting blood glucose	☐	☐	☐
30 minute blood glucose	☐	☐	☐
1 hour blood glucose	☐	☐	☐
2 hour blood glucose	☐	☐	☐
3 hour blood glucose	☐	☐	☐
Fasting urine	☐	☐	☐
30 minute urine	☐	☐	☐
1 hour urine	☐	☐	☐
2 hour urine	☐	☐	☐
3 hour urine	☐	☐	☐

REMARKS_____

symptom, it was scored in only four categories (Fig. 1).

Pain with Tooth Percussion: Soreness of the teeth when the jaws are brought together has been reported as a symptom of diabetes mellitus[12, 13, 15, 16, 17, 18, 19] so an attempt was made to record the patient's story in one of five groups (Fig. 1).

Dry Sockets: The evidence from an appraisal of the literature is that alveolar osteitis is a frequent sequelum of tooth extraction in the diabetic patient.[20, 21] There is even some evidence[22] that dry sockets are twice as frequent in diabetic as against nondiabetic patients. Because of the difficulty of establishing whether dry sockets did or did not occur, only a three-point scale was used (Fig. 1).

CLINICAL (ORAL) EXAMINATION

Evidence has already been presented to show that lesions identified with diabetes mellitus occur exclusively or at least predominantly in certain oral zones.[2] These include: (1) *lips*, (2) *gingiva*, (3) *tongue*, and (4) *teeth*. Accordingly, these special areas were investigated in this study.

Lips: Dryness of the lips has been mentioned in the literature[10] though no figures are cited. In this study, this particular sign was recorded on a four-point scale (Fig. 1).

Gingiva: Mention has been made in the literature of the association of gingival pathosis and diabetes mellitus. Therefore, in this investigation, attention was directed to: (1) *gingival stippling*, (2) *gingival bleeding*, (3) *gingival hue*, and (4) *pocket formation*.

Gingival Stippling: Normally, the gingiva is characterized by stippling. The absence of such stippling suggests the presence of inflammation and edema. This finding has been related to diabetes mellitus.[11, 12, 14, 15, 16, 17, 18, 20] Only one group[23] has indicated a specific figure—15 percent occurrence of spongy gingiva. In this study the gingiva was graded on a four-point scale (Fig. 1).

Gingival Bleeding: The presence of gingival bleeding and diabetes mellitus has been mentioned.[11, 12, 14, 16, 17, 19, 20] Only one group have cited a prevalence and that is 32 percent.[23] This finding was gauged according to the Roth classification[24] on a five-point scale (Fig. 1).

Gingival Hue: Ordinarily the gingiva is characterized by a coral pink hue. With inflammation the color deepens. Violaceous gingiva has been reported as part of the diabetic picture.[17, 18] One group[23] report gingival color change in 92 percent of their juvenile series. In this study the findings were scored 0, 1, and 2 (Fig. 1).

Pocket Formation: Under physiologic conditions, the base of the gingival sulcus is at the cementoenamel junction, and the sulcus is generally not more than one mm. in depth. One group of investigators[23] cited a prevalence of 89 percent of diabetic children with periodontal pockets. In this report, pocket formation was scored on an eight-point scale (Fig. 1) to allow recording of shallow and deep absolute and relative pockets and combinations of absolute and relative pocket formations.

Modification of the P. M. A. Index: As a check of the periodontal status, the Parfitt modification of the P. M. A. Index[25] was utilized as shown in Fig. 1.

Tongue: The dorsum of the tongue was examined in some detail with particular emphasis as to: (1) *degree of smoothness*, (2) *presence of geographic tongue*, (3) *color*, (4) *size*, and (5) *degree of fissuring*.

Smoothness of Tongue: Normally, the dorsum of the tongue is studded with filiform and fungiform papillae. Only one investigator[10] observed areas of denudation on the dorsum of the tongue which he felt were associated with diabetes mellitus. The status of the dorsum of the tongue was graded in a four-point scale (Fig. 1).

Geographic Tongue: The dorsum of the tongue, under normal conditions, contains no patchy areas of denudation. Their presence was graded in this report from 0 through 2 (Fig. 1).

Red Tongue: In health the dorsum of the tongue is pink in color. Two investigators[10, 20] simply point out an association between diabetes mellitus and lingual erythema. Gradations in lingual hue were charted on a three-point scale (Fig. 1).

Tongue Size: Ordinarily, the sides of the tongue in a dentulous patient are smooth. The presence of so-called marginal indentations is suggestive evidence of tongue enlargement. Two investigators[10, 20] have claimed macroglossia as a diabetic finding. In this investigation, tongue size was graded 0, 1, or 2 (Fig. 1).

Fissures on the Tongue: Ordinarily, there is but one fissure present in the sagittal line of the tongue. However, in some instances, other grooves and fissures may be observed. Three groups of investigators[10, 18, 20] have reported fissuring of the tongue as a sign of diabetes mellitus. In this report fissures on the tongue were scored on a three-point scale (Fig. 1).

Teeth: The three most common dental findings attributed to diabetes mellitus are: (1) *increased tooth mobility*, (2) *increased tooth loss*, and (3) *calculus*.

Increased Tooth Mobility: Usually, the teeth are only minimally mobile, if at all. A number of investigators[12, 13, 15, 16, 19] have reported loosening of the teeth as judged clinically simply by checking mobility by finger force. This was also done as part of this study and reported on a three-point scale as shown in Fig. 1.

Increased Tooth Loss: Two reports are available[14, 20] indicating that tooth loss in diabetic patients is greater than in a nondiabetic population. In this re-

port the total number of teeth present was recorded for every patient (Fig. 1).

Calculus: The general statement appears in the literature that the diabetic patient usually shows little or no calculus. In this investigation calculus was scored on an eight-point scale as shown in Fig. 1.

ROENTGENOGRAPHIC EXAMINATION

The two most discussed roentgenographic findings related to diabetes mellitus are: (1) *alveolar bone loss,* and (2) *marginal widening of the periodontal membrane.*

Alveolar Bone Loss: A number of different techniques have been used for measuring alveolar bone height. Sheppard[27] measured the amount of resorption of the dental alveolar septum by dividing the root length into degrees of 0 to 10. Five indicated that one-half of the alveolar support of the tooth was missing. Miller and Seidler[28] utilized a modification of Sheppard's method of roentgenographic analysis. The amount of bone resorption was scored by dividing the root length into degrees of 0 to 5 with 3 indicating one-half the alveolar support. Miller and Pelzer[29] proposed a more elaborate roentgenographic classification. They graded the interdental septum and alveolar crest in five categories. Group 1 was described as being normal and characterized by uniform density of the septum with the lamina dura extending over the septal crest. The second type demonstrated marginal widening of the periodontal membrane and associated narrowing or thinning of the lamina dura. Type 3 simply represented the first type except that the alveolar crest was present at a more apical level. The fourth type was characterized by the absence of the lamina dura over the septal crest. The fifth and last category indicated rapid bone destruction with the absence of the lamina dura over the septal crest and increased roentgenolucency toward the crest. It is obvious from this brief account that there are various methods of scoring alveolar bone status. All of them have advantages and disadvantages. For purposes of this study a six-point scoring system was utilized as shown in Fig. 1.

Marginal Widening of the Periodontal Membrane: Under physiologic conditions, the periapical roentgenogram discloses a roentgenolucent thin line extending completely about the root of the tooth. Occasionally, this thin line which indicates the position and extent of the periodontal membrane may be widened. Membrane widening in this study was graded on a three-point scale (Fig. 1).

LABORATORY STUDIES

A one-dose three-hour oral glucose tolerance test was performed on each patient according to the method of Folin and Wu,[30] Nelson,[31] and Somogyi.[32, 33] Simultaneous urinalyses were checked for reducing substances (Clinitest) at the fasting, 30 and 60 minute, and 2 and 3 hour periods (Fig. 1). No dietary instructions were given to the patient prior to the laboratory examination except for fasting from the night before the test date.

There is no question but that there is still considerable debate as to the laboratory criteria for diabetes mellitus. No one will take issue with the glucose tolerance pattern which is unequivocally normal and one which is undeniably that of a diabetic patient. The arguments center more about the intermediate and infinite gradations between obvious normality and clearcut diabetes mellitus. Many of the arguments have been recently reviewed by Unger.[34] For purposes of this study patients were grouped into four categories:

Group I. Individuals showing a *decrease* in glucose tolerance as evidenced by a blood true glucose of 150 or more mg. percent during the three-hour test *and* a failure to return to below 100 mg. percent at the two-hour period. According to many investigators, such individuals are regarded as suffering with diabetes mellitus.

Group II. Individuals characterized by a mild decrease in glucose tolerance as evidenced by a rise in true glucose to above 150 mg. percent *or* a failure to return to below 100 mg. percent at the two-hour period. According to some investigators, these individuals are regarded as suffering with prediabetes mellitus.

Group III. Individuals characterized by a glucose tolerance test in which the fasting true glucose is below 100 mg. percent, the level does not rise above 150 mg. percent at any time, and the blood glucose returns to below 100 mg. percent in two hours. These individuals, according to most investigators, are declared as being normal in terms of carbohydrate metabolism.

Group IV. Individuals showing an *increase* in tolerance to glucose and being characterized by a glucose tolerance pattern in which the true glucose in the blood never exceeds 100 mg. percent in any of the samples. These individuals are frequently referred to as demonstrating a *flat* glucose tolerance pattern.

The significance of these various tolerance patterns will be considered later in this report.

RESULTS

The findings of this study will be considered in *five* categories: (1) *general characteristics,* (2) *symptomatology* as derived from the history, (3) *oral signs,* the result of the clinical examination, (4) *roentgenographic findings,* and (5) *laboratory data.*

General Characteristics: A number of interesting aspects of the study are worthy of consideration including: (1) *patient classi-*

fication on the basis of the glucose tolerance pattern, (2) *age*, (3) *sex*, (4) *knowledge by the patient regarding possible diabetic status*, (5) *reason for admission* to the dental clinic, and (6) *family history* of diabetes mellitus.

Patient Classification: Table 1 shows the number and percentage of patients classified in the various groups. It is of interest that approximately one out of four of these individuals showed evidence of moderate to marked decrease in glucose tolerance. An additional 17 percent (Group II) demonstrated a *marginal* decrease in carbohydrate metabolism. Of particular note is the fact that only approximately four out of ten individuals could be regarded as showing glucose tolerance patterns unequivocally normal. Finally, mention should be made of the rather important observation that one out of five individuals demonstrated a glucose tolerance curve which was either flat or actually inverted and a mirror image of the type of curve observed in diabetes mellitus. The finding of 26 percent of the individuals with an abnormal decrease in glucose tolerance is supported by the observation[35] made in an earlier study of 100 patients referred for full mouth extractions.

TABLE 1—Patient Classification

Groups	*Number and percentage of patients*
I	26
II	17
III	37
IV	20
Total	100

Age Distribution: Table 2 shows the age distribution in decades compared with the glucose tolerance pattern. Mention should first be made of the fact that the total number of patients approaches a typical unimodal curve with slight skewing. This is supported by the observation that only one individual in the group was less than 10 years of age whereas seven were above the age of 60. However, 27 percent of the individuals were in the 30-39 year group and 19 percent each were in the 20-29 and 40-49 year group. Further examination of the chart discloses that the percentage of individuals with moderate to marked decreases in sugar tolerance increased with the age group. This can be observed in the Group I column where one notes a percentage of 11 in the 10-19 year group and a progressive increase to 22 percent in the 30-39 year group and, finally, 50 percent in the 60-69 year group. This progressive pattern is not evident in any of the other groups.

Sex Distribution: Table 3 outlines the

TABLE 2—Age Distribution

Age group	*I*		*II*		*III*		*IV*		*Total number of patients*	*Total percentage*
	No.	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*		
0– 9	—	—	—	—	1	100	—	—	1	100
10–19	1	11	2	23	3	33	3	33	9	100
20–29	2	11	3	15	11	58	3	16	19	100
30–39	6	22	2	7	11	41	8	30	27	100
40–49	6	32	4	21	4	21	5	26	19	100
50–59	7	40	5	30	4	24	1	6	17	100
60–69	4	50	1	12	3	38	—	—	8	100
Total	26		17		37		20		100	

TABLE 3—Sex Distribution

Group	Number of male patients	Percentage	Number of female patients	Percentage	Total number of patients	Total percentage
I	5	19	21	81	26	100
II	6	36	11	64	17	100
III	11	30	26	70	37	100
IV	—	—	20	100	20	100
Total	22		78		100	

patients according to sex and glucose tolerance pattern. It can be observed that four out of every five patients showing a moderate to marked decrease in glucose tolerance proved to be female. Of particular interest is the fact that all of the twenty patients who demonstrated flat to inverted tolerance patterns were also female.

Diabetic Status: Each patient was questioned as to whether he or she was or was not diabetic. Table 4 summarizes the information. One can observe that only 8 percent of those with a moderate to marked decrease in glucose tolerance reported that they had been told that they were suffering with diabetes mellitus. The percentage, as shown in Table 4, decreases to 6 percent in those with so-called marginal decreases in tolerance. Not one of the 57 individuals with unequivocally normal to flat or inverted curves had ever been told that he was suffering with diabetes mellitus. Actually, as shown in this chart, only two individuals in the entire series were known diabetics.

Reason for Admission: It should be recalled that all of the patients in this study visited the Dental Clinic voluntarily. At random some of the individuals were referred to the Oral Medicine Clinic for this study. Table 5 summarizes the reasons why these patients sought dental advice. Approximately one-half (49 percent) simply came to the dental school for routine examination. Of this group who were completely asymptomatic, about one-half (25 percent) demonstrated perfectly normal glucose tolerance patterns. One out of six (14 percent) patients reporting for routine examination proved to have glucose tolerance curves highly suggestive of diabetes mellitus. Mention should be made of the rather interesting observation that, of the 3 percent of the patients who visited the school because of loose teeth, all of them demonstrated either a marginal or marked decrease in glucose tolerance. Finally, three of the patients who were admitted to the study had heard of the diabetic survey and for that reason reported. It can be observed

TABLE 4—Diabetic Status

Group	Yes		No		Total number of patients	Total percentage
	No.	%	No.	%		
I	2	8	24	92	26	100
II	1	6	16	94	17	100
III	0	0	37	100	37	100
IV	0	0	20	100	20	100

TABLE 5—Reason for Admission

	I		II		III		IV		Total number of patients	Total percentage
	No.	%	No.	%	No.	%	No.	%		
Routine check	7	14	10	20	20	41	12	25	49	100
Loose teeth	2	67	1	33	0	0	0	0	3	100
Sore gums	4	33	3	25	3	25	2	17	12	100
Sore teeth	5	33	1	7	7	47	2	13	15	100
Sore tongue	0	0	1	50	0	0	1	50	2	100
Sore cheek	3	60	0	0	2	40	0	0	5	100
Diabetic survey	1	33	0	0	1	33	1	34	3	100
Teeth extracted	4	36	1	9	4	36	2	19	11	100
Total	26		17		37		20		100	

TABLE 6—Family History of Diabetes Mellitus

Group	Negative		Positive		Total number of patients	Total percentage
	No.	%	No.	%		
I	20	77	6	23	26	100
II	16	94	1	6	17	100
III	27	73	10	27	37	100
IV	15	75	5	25	20	100

TABLE 7—Boils

Group	Absent		Occasional		Constantly		Total number of patients	Total percentage
	No.	%	No.	%	No.	%		
I	20	77	5	19	1	4	26	100
II	11	65	6	35	—	—	17	100
III	30	81	7	19	—	—	37	100
IV	15	75	5	25	—	—	20	100

TABLE 8—Appetite

Group	Same		Increased		Decreased		Total number of patients	Total percentage
	No.	%	No.	%	No.	%		
I	16	62	6	23	4	15	26	100
II	13	76	2	12	2	12	17	100
III	28	76	6	16	3	8	37	100
IV	16	80	3	15	1	5	20	100

that one proved to show moderate to marked decrease while another demonstrated moderate to marked increase in glucose tolerance. The remaining one showed a normal glucose tolerance curve.

Family History: It is interesting to observe, as shown in Table 6, that only one in four of the persons with moderate to marked decrease in glucose tolerance reported a positive family history of diabetes mellitus. This same positive history was obtained in one out of every four of those persons showing a diametrically opposite type of glucose curve (flat to inverted). It would appear, from these data, that the frequently described significance of family history is not particularly helpful in dealing with dental patients who, if they have diabetes mellitus, are suffering with the very earliest manifestations.

In a later section of this report, special attention will be directed to those persons with normal glucose tolerance patterns and positive family history.

Symptomatology: In the main, those symptoms were investigated which could be elicited by a dental examiner. These were divided into two categories: (1) *extraoral symptoms*, and (2) *intraoral symptoms*.

The extraoral symptoms items which were studied were those frequently described as being commonly associated with diabetes mellitus. Accordingly, the following were recorded: (1) *boils*, (2) *appetite*, (3) *fluid intake*, (4) *fluid excretion*, and (5) *weight change*.

Boils: Table 7 summarizes the data elicited from the patients regarding the occasional or constant appearance of furuncles. From this chart it appears that there is no relationship between furunculosis and the glucose tolerance pattern. The only possibly significant observation which can be made is that the one individual in the entire series who reported constant furunculosis proved to have a glucose tolerance pattern suggestive of diabetes mellitus.

Appetite: Table 8 shows the percentage of individuals in each of the groups who reported unchanged, increased, or decreased appetite during the preceding six-month period. One observes in the first column in Table 8 that the number of individuals with unchanged appetite increased as one progressed from those with decreased tolerance to those with increased tolerance. Apparently, this progression is largely the result of the rather orderly decrease in appetite noted in Table 8. For example, 15 percent of the individuals demonstrated decreased glucose tolerance whereas only one-third as many reported decreased appetite in the group with moderate to marked increased glucose tolerance.

Fluid Intake: Table 9 summarizes the data regarding changes in fluid intake during the preceding six-month period. Mention should be made of the fact that the two groups reporting the most obvious increase in fluid intake were those at the extremes (those with moderate to marked increase and decrease in tolerance). This is shown by a percentage of 19 in those in Group I and 25 percent of those in Group IV.

Fluid Excretion: Table 10 summarizes the information regarding alterations in urinary excretion during the preceding six-month period. One can observe that the greatest number of individuals showing increased excretion were in Group I.

Weight Change: Table 11 summarizes the information obtained from the patients regarding weight change during the preceding six-month period. The greatest occurrence of weight loss was observed in the patients with decreased tolerance (Groups I and II).

Mention has been made in the preceding report[1] that the most common subjective complaints reported are: (1) *dry mouth*, (2) *burning mouth*, (3) *tender gingiva*, (4) *pain with tooth percussion*, and (5) *dry sockets*. Accordingly, these same items have been included in the present study.

Dry Mouth: According to Table 12 it appears that occasional to constant dry

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TABLE 9—Fluid Intake

Group	Same No.	Same %	Increased No.	Increased %	Decreased No.	Decreased %	Total number of patients	Total percentage
I	20	77	5	19	1	4	26	100
II	15	88	2	12	0	0	17	100
III	32	86	4	11	1	3	37	100
IV	15	75	5	25	0	0	20	100

mouth occurs much more commonly in those with decreased tolerance (54 percent) than in any other group. Mention should be made of the fact that, of the three patients who reported constant dry mouth, two demonstrated tolerance patterns which were markedly decreased and the remaining one proved to be at the other extreme.

Burning Mouth: The observations made regarding dry mouth appear to hold also for the burning complaint (Table 13). It can be observed that this symptom was reported much more frequently (five to sevenfold) in those with decreased tolerance than in any other group.

Tender Gingiva: An examination of Table 14 discloses that the occasional appearance of tenderness of the gingiva seemed to occur most often in those with a decrease in sugar tolerance as shown by 54 percent in Group I. This is underscored by the observation that one out of three in Group I reported the most (occasional to constant) gingival tenderness.

Pain with Tooth Percussion: The evidence appears to be (Table 15) that sensitivity to tooth percussion occurs much more frequently (two to fourfold) in the Group I category than in any of the others.

Dry Sockets: The data from Table 16 indicates no obvious relationship between the occurrence of alveolar osteitis and the glucose tolerance pattern.

CLINICAL (ORAL) EXAMINATIONS

In order to quantitate the *signs* with which diabetic as well as nondiabetic patients complain, an analysis was made of the following items: (1) *dryness of the lips,* (2) *gingival stippling,* (3) *gingival bleeding,* (4) *gingival hue,* (5) *pocket formation,* (6) *P.M.A. Index,* (7) *smooth tongue,* (8) *geographic tongue,* (9) *tongue color,* (10) *tongue size,* (11) *tongue fissures,* (12) *tooth mobility,* (13) *number of teeth,* and (14) *calculus.*

Dryness of the Lips: According to Table 17, the occurrence of dryness of the lips seems to be more prominent in the Group I category with a finding of approximately twice as many individuals with this complaint in this group as compared to any

TABLE 10—Fluid Excretion

Group	Normal No.	Normal %	Increased No.	Increased %	Decreased No.	Decreased %	Unknown No.	Unknown %	Total number of patients	Total percentage
I	15	58	10	38	0	0	1	4	26	100
II	13	76	4	24	0	0	0	0	17	100
III	28	76	9	24	0	0	0	0	37	100
IV	15	75	5	25	0	0	0	0	20	100

TABLE 11—Weight Change

Group	Same No.	Same %	Increased No.	Increased %	Decreased No.	Decreased %	Total number of patients	Total percentage
I	12	46	6	23	8	31	26	100
II	6	35	4	24	7	41	17	100
III	23	62	8	22	6	16	37	100
IV	12	60	6	30	2	10	20	100

TABLE 12—Dry Mouth

Group	No dry mouth No.	No dry mouth %	Occasional dry mouth No.	Occasional dry mouth %	Constant dry mouth No.	Constant dry mouth %	Total number of patients	Total percentage
I	12	46	12	46	2	8	26	100
II	12	71	5	29	0	0	17	100
III	26	70	11	30	0	0	37	100
IV	14	70	5	25	1	5	20	100

TABLE 13—Burning Mouth

Group	Absent No.	Absent %	Occasional No.	Occasional %	Total number of patients	Total percentage
I	17	65	9	35	26	100
II	16	94	1	6	17	100
III	34	92	3	8	37	100
IV	19	95	1	5	20	100

TABLE 14—Tender Gingiva

Group	Absent No.	Absent %	Occasional No.	Occasional %	Constant No.	Constant %	Total number of patients	Total percentage
I	10	38	14	54	2	8	26	100
II	9	53	7	41	1	6	17	100
III	25	68	9	24	3	8	37	100
IV	11	55	7	35	2	10	20	100

TABLE 15—Pain with Tooth Percussion

Group	None No.	None %	Slight No.	Slight %	Severe No.	Severe %	Total number of patients	Total percentage
I	16	76	4	19	1	5	21	100
II	15	94	1	6	0	0	16	100
III	30	88	4	12	0	0	34	100
IV	17	89	2	11	0	0	19	100

TABLE 16—Dry Sockets

Group	Absent No.	Absent %	Present No.	Present %	Total number of patients	Total percentage
I	21	81	5	19	26	100
II	13	76	4	24	17	100
III	25	68	12	32	37	100
IV	15	75	5	25	20	100

TABLE 17—Dryness of Lips

Group	No No.	No %	Mild No.	Mild %	Severe No.	Severe %	Total number of patients	Total percentage
I	18	69	6	23	2	8	26	100
II	15	88	2	12	0	0	17	100
III	31	84	6	16	0	0	37	100
IV	18	90	2	10	0	0	20	100

of the other categories. Attention should be directed to the fact that, in the two individuals in the entire series who showed severe dryness of the lips, both proved to have also decreased tolerance to glucose.

Gingival Stippling: According to Table 18 it appears that the greatest percentages of normal gingival stippling occurred in the two extreme categories (Groups I and IV). In the case of slight loss of gingival stippling, there seems to be no particular rhyme or reason except that the slight disappearance of stippling occurs rarely in the Group IV patients. The most interesting observation that can be made about Table 18 is that, of the four individuals who showed severe loss of gingival stippling, all of them demonstrated decreased glucose tolerance.

Gingival Bleeding: It can be observed in Table 19 that toothbrush bleeding increased with increasing tolerance to glucose. On the other hand, so-called spontaneous bleeding showed a reverse pattern with decrease in spontaneous bleeding associated with increased carbohydrate tolerance.

Gingival Hue: In general, there appears to be no relationship between gingival hue and carbohydrate metabolism as measured in this experiment. The only finding worthy of mention (Table 20) is the fact that obvious alterations in gingival hue occur largely (two to fourfold) in those individuals with decreased glucose tolerance (Groups I and II).

Pocket Formation: The pattern of pocket formation is outlined in Table 21. One can observe the least number of individuals with no pocket formation in Group I and the greatest percentage in Group IV. By and large, this appears to be due to the presence of absolute pocket formation as shown in the third column in Table 21. It can be observed that four out of five individuals in Group I demonstrated absolute pocket formation. In contrast, one in two showed the same findings in Group IV. Groups II and III reveal intermediate scores. It is worthy of mention that in no case did those individuals showing extremes in glucose tolerance demonstrate relative pocket formation.

TABLE 18—Gingival Stippling

Group	Normal No.	%	Slight loss No.	%	Moderate loss No.	%	Severe loss No.	%	Total number of patients	Total percentage
I	11	52	7	33	1	5	2	10	21	100
II	7	41	7	41	1	6	2	12	17	100
III	13	40	16	48	4	12	0	0	33	100
IV	14	78	1	6	3	16	0	0	18	100

TABLE 19—Gingival Bleeding

Group	No No.	%	With tooth brushing No.	%	With biting apple, etc. No.	%	Spontaneous No.	%	Total number of patients	Total percentage
I	13	63	5	21	0	0	3	16	21	100
II	11	64	4	24	1	6	1	6	17	100
III	15	46	15	46	2	5	1	3	33	100
IV	6	33	11	61	1	6	0	0	18	100

TABLE 20—Gingival Hue

Group	Coral pink No.	%	Red No.	%	Blue to purple No.	%	Total number of patients	Total percentage
I	20	76	3	12	3	12	26	100
II	11	65	4	23	2	12	17	100
III	27	73	9	24	1	3	37	100
IV	16	80	3	15	1	5	20	100

P. M. A. Index: By and large, it can be observed from Table 22 that the P. M. A. index was poorest in Group I and best in Group IV. However, no significant pattern can be shown on the basis of *severity* in periodontal pathosis.

Smooth Tongue: The evidence, as shown in Table 23, indicates no correlation between the tolerance pattern and the smoothness of the dorsum of the tongue.

Geographic Tongue: Table 24 suggests that there is no significant relationship between migratory areas of denudation on the dorsum of the tongue and carbohydrate metabolism.

Tongue Color: Mention should be made of the fact that mild redness of the tongue occurred principally in those showing extremes in tolerance (Table 25). For example, it can be shown that one out of five in Group I and one out of six in Group IV demonstrated alterations in tongue color in contrast to practically none in the intermediate categories.

Tongue Size: It appears, from Table 26, that there is a progressive decrease in mar-

TABLE 21—Pocket Formation

Group	Normal No.	%	Absolute pockets No.	%	Relative pockets No.	%	Absolute & relative pockets No.	%	Total number of patients	Total percentage
I	2	9	17	81	0	0	2	10	21	100
II	3	19	9	56	1	6	3	19	16	100
III	11	32	20	59	2	6	1	3	34	100
IV	8	44	8	44	0	0	2	12	18	100

TABLE 22—P. M. A. Index

Group	0-.9 No.	%	1-1.9 No.	%	2-2.9 No.	%	3-3.9 No.	%	4-4.9 No.	%	Total number of patients	Total percentage
I	7	33	5	24	5	24	3	14	1	5	21	100
II	6	37	3	19	3	19	3	19	1	6	16	100
III	15	44	9	26	3	9	4	12	3	9	34	100
IV	10	55	3	17	2	11	3	17	0	0	18	100

TABLE 23—Smooth Tongue

Group	Normal No.	%	Slight No.	%	Moderate No.	%	Generalized No.	%	Total number of patients	Total percentage
I	21	81	4	15	1	4	0	0	26	100
II	12	70	4	24	1	6	0	0	17	100
III	30	81	7	19	0	0	0	0	37	100
IV	17	85	2	10	1	5	0	0	20	100

ginal indentations with increase in glucose tolerance. One observes that approximately one out of four patients in Group I show marginal indentations in contrast to one in ten in Group IV.

Tongue Fissures: According to Table 27, there appears to be no significant differences in the presence of tongue fissures in the various groups.

Tooth Mobility: It is interesting to observe, as shown in Table 28, a logical progression. For example, almost one-half of the Group I patients showed tooth mobility greater than 1 mm. In contrast, only one in twenty of those with increased tolerance demonstrated this same finding. Groups II and III are shown to have intermediate scores.

Number of Teeth: An examination of Table 29 shows the number of teeth observed in the four groups. It can be seen that approximately two-thirds of Group I patients had 8.5 to 27.4 teeth. In contrast, those with unequivocally normal tolerance patterns demonstrated 12.9 to 29.5 teeth.

TABLE 24—Geographic Tongue

Group	No		Mild		Severe		Total number of patients	Total percentage
	No.	%	No.	%	No.	%		
I	25	96	1	4	0	0	26	100
II	17	100	0	0	0	0	17	100
III	36	97	1	3	0	0	37	100
IV	19	95	1	5	0	0	20	100

TABLE 25—Tongue Color

Group	Pink		Mild red		Bright red		Total number of patients	Total percentage
	No.	%	No.	%	No.	%		
I	21	81	5	19	0	0	26	100
II	17	100	0	0	0	0	17	100
III	35	95	2	5	0	0	37	100
IV	17	85	3	15	0	0	20	100

TABLE 26—Tongue Size

Group	No marginal indentations		Slight marginal indentations		Marked marginal indentations		Total number of patients	Total percentage
	No.	%	No.	%	No.	%		
I	20	77	6	23	0	0	26	100
II	14	82	3	18	0	0	17	100
III	29	78	7	19	1	3	37	100
IV	18	90	2	10	0	0	20	100

TABLE 27—Tongue Fissures

Group	None		Slight		Severe		Total number of patients	Total percentage
	No.	%	No.	%	No.	%		
I	24	92	2	8	0	0	26	100
II	16	94	1	6	0	0	17	100
III	32	86	4	11	1	3	37	100
IV	18	90	1	5	1	5	20	100

TABLE 28—Tooth Mobility

Group	None		<1 mm.		>1 mm.		Total number of patients	Total percentage
	No.	%	No.	%	No.	%		
I	5	24	6	29	10	47	21	100
II	6	38	6	38	4	24	16	100
III	21	62	9	26	4	12	34	100
IV	11	61	6	33	1	6	18	100

Calculus: It is noteworthy to observe (Table 30) that the absence of calculus increased with increasing tolerance. One notes that one in four patients in Group I showed no calculus in contrast to almost one in two in Group IV. Also, it appears that supragingival calculus occurred much less frequently in Group IV patients than in Group I. For example, approximately one in three patients in Group I demonstrated supragingival calculus in contrast to one in six in Group IV. This same progression was observed with regard to the presence of both supra- and subgingival calculus.

ROENTGENOGRAPHIC FINDINGS

The two most discussed roentgenographic findings associated with diabetes mellitus are: (1) *alveolar bone loss,* and (2) *marginal widening of the periodontal membrane.* These are considered here in separate sections.

Alveolar Bone Loss: Mention should be made of the fact (Table 31) that normal septal crests occurred least in those patients showing extremes in glucose tolerance. One observes that not one patient in Group I showed normal septal crest formation and only one in twenty in Group IV. It appears that there is a relationship between glucose tolerance and this particular roentgenographic finding.

TABLE 29—Number of Teeth

Group	*Mean*	*Standard deviation*
I	18.19	9.70
II	23.05	7.94
III	21.21	8.32
IV	21.55	9.21

Widening of the Periodontal Membrane: It is interesting to observe (Table 32) that two-thirds of the Group I patients showed widening. One can observe in this chart that the figures decrease with increasing glucose tolerance.

LABORATORY FINDINGS

The incidence of diabetes mellitus in various epidemiologic studies varies with the criteria used to establish a diagnosis of diabetes mellitus. In some of the reports only urinalysis was used as the screening technique. On the other hand, in other investigations fasting and two-hour postprandial blood sugars have been employed. In addition, the levelt of sugar recognized as diagnostic has varied with different investigators. It was thought interesting to analyze some of the already-employed criteria to determine how many diabetic patients would have been noted had some of these standards been utilized.

TABLE 30—Calculus

	I		*II*		*III*		*IV*	
	No.	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*
Absence	5	24	5	31	14	40	8	44
Slight supragingival	5	24	2	13	5	14	3	16
Moderate supragingival	1	5	1	6	1	3	0	0
Slight subgingival	0	0	0	0	0	0	0	0
Moderate subgingival	3	14	1	6	0	0	1	6
Slight supra- and subgingival	3	14	2	13	6	17	1	6
Moderate supra- and subgingival	4	19	2	13	3	9	1	6
Marked supra- and subgingival	0	0	3	18	5	17	4	22
Total number of patients	21		16		34		18	
Total percentage		100		100		100		100

TABLE 31—Alveolar Bone Loss

	I		II		III		IV	
	No.	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*
Normal septal crest	0	0	2	12	8	24	1	5
Minimal alveolar bone loss	4	19	2	12	8	24	9	50
Minimal to moderate alveolar bone loss	6	29	6	38	6	18	3	17
One-half the alveolar support missing	3	14	3	19	7	22	3	17
Moderate to severe alveolar bone loss	7	33	3	19	4	12	2	11
No alveolar support	1	5	0	0	0	0	0	0
Total number patients	21		16		33		18	
Total percentage		100		100		100		100

Table 33 shows that, in this study, had glycosuria been utilized exclusively as a screening procedure, then one out of three individuals in Group I would have been detected. Put another way, two out of three of these people would never have been pursued with other laboratory studies to determine the absence or presence of diabetes mellitus. It is also of some interest to note that, had a fasting blood glucose been utilized as screening yardstick, then only one in four of the patients would have been suspected of having diabetes mellitus. This is in contrast to the findings utilizing the true glucose tolerance procedure. The reason for the 96 percent values in Group I is due to the one controlled diabetic individual who did not meet the usual laboratory criteria but who obviously had diabetes mellitus because of past history and therapy.

DISCUSSION

The results will be considered in five categories: (1) *general characteristics*, (2) *symptoms*, (3) *signs*, (4) *roentgenographic findings*, and (5) *laboratory data*.

General Characteristics: A number of items of a general informational type have been reported in the section entitled Results. This information will now be considered in *six* categories: (1) *patient classification*, (2) *age*, (3) *sex*, (4) *knowledge on the part of the patient regarding possible diabetes mellitus*, (5) *reason for admission*, and (6) *familial history of diabetes mellitus*.

Patient Classification: There is still considerable controversy as to the limits of the *normal* glucose tolerance test. In this experiment 37 percent of the glucose toler-

TABLE 32—Widening of Periodontal Membrane

Group	*No marginal widening*		*Minimal marginal widening*		*Severe widening*		*Total number of patients*	*Total percentage*
	No.	*%*	*No.*	*%*	*No.*	*%*		
I	8	38	12	57	1	5	21	100
II	7	44	8	50	1	6	16	100
III	20	61	12	36	1	3	33	100
IV	12	67	6	33	0	0	18	100

TABLE 33—Detection of Diabetes Mellitus by Different Laboratory Criteria

	I				II				III				IV			
	Positive		*Negative*		*Positive*		*Negative*		*Positive*		*Negative*		*Positive*		*Negative*	
	No.	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*
Fasting glycosuria	9	35	17	65	0	0	17	100	0	0	37	100	0	0	20	100
Fasting blood glucose >100 mg. percent	6	23	20	77	0	0	17	100	0	0	37	100	0	0	20	100
Any blood glucose >150 mg. percent	25	96	1	4	5	29	12	71	0	0	37	100	0	0	20	100
Two-hour blood glucose >100 mg. percent	26	100	0	0	12	71	5	29	0	0	37	100	0	0	20	100
Any blood glucose >150 and two-hour >100	25	96	1	4	0	0	17	100	0	0	37	100	0	0	20	100

ance patterns could be placed, without any question, in this category. There is, however, reasonable agreement as to what constitutes *classical* diabetes mellitus. However, it is impossible to establish hard and fast rules for the line of demarcation between typical diabetes mellitus, prediabetes, and normality.

According to the criteria established for this study, 26 percent of the patients showed a glucose tolerance pattern indicative of diabetes mellitus (Table 1). Seventeen percent of the individuals displayed a tolerance pattern regarded by many investigators as evidence of prediabetes mellitus. Finally, in this survey of cases, one out of five individuals demonstrated a tolerance pattern regarded by most of the authorities as being flat or inverted and indicating increased tolerance to glucose.

The groups which lend themselves to the greatest controversy are II and IV. However, it was felt advisable to so categorize the subjects in this study because, as we shall see, many of the findings in Group II resemble more closely the clinical picture of patients in Group I than in Group III. Justification for Group IV will be apparent when one notes that, in a significant number of instances, the findings in Group IV patients more closely parallel Group I than Groups II or III.

It is difficult to draw conclusions from these observations and compare them with the literature reports. Rochon and Moran[36] reported 1.2 percent previously unrecognized diabetic patients in their Clinitest analysis of 3,122 dental patients. In this survey of 100 dental patients studied by means of true glucose tolerance tests, 26 percent of the cases showed decreased sugar tolerance of the magnitude regarded by many as indicative of diabetes mellitus. In other words, if a comparison can be made and this can only be done with caution, then detection increased *twenty-one-fold* as a result of the more elaborate laboratory testing procedures. The possible validity of the parallelism is heightened by the observations reported previously.[1] It should be recalled that the detection among the general population proved to be approximately 0.2 percent by urinalysis alone.[37] The use of urinalysis plus blood sugar raised the percentage to 5 percent.[38] This indicates a *twenty-five-fold* increase. Obviously more such studies must be carried out to confirm or negate these limited observations. Actually, one such project is presently being conducted.[39]

Age Distribution: An examination of Table 2 discloses the increasing number of individuals in Group I with advancing age. This observation is consistent with the reports in the literature.[40,41,42] However, this fact complicates matters for it is a fact that, presumably in healthy persons, periodontal pathosis increases with age. Therefore, in subsequent discussions, the data will be viewed with respect to age as well as other factors.

Sex Distribution: The evidence, as shown

TABLE 34—Mean Number of Teeth

Age group	*Group I*	*Group III*
<30	20.66	23.60
30–39	23.83	19.09
40–49	20.50	25.00
50–59	15.00	19.25
60–69	10.00	14.66

in Table 3, indicates that the female demonstrated the widest fluctuations in glucose tolerance as evidenced by the fact that the greatest percentage of individuals in Groups I and IV were of the female sex. As a matter of fact, it is rather significant that every patient showing a flat to inverted glucose tolerance pattern proved to be female.

Diabetic Status: It appears from Table 4 that, from a dental standpoint, one cannot rely upon the patient's statements as to whether he or she is or is not suffering from diabetes mellitus. One observes from Table 4 that 92 percent of the individuals who proved to show fairly definite evidence of decreased tolerance to carbohydrates were not aware of this finding. The point to be gleaned from this observation is that laboratory tests must be done in order to establish the existence of abnormal carbohydrate metabolism.

Rochon and Moran[36] observed a prevalence of 0.1 percent of previously known diabetic patients in their survey. In this report, 2 percent known diabetic patients was observed making the difference *twenty-fold*. The entire explanation for this great difference may not be forthcoming. However, one can explain the discrepancy, at least in part, by the following items: (1) Rochon and Moran used only urinalysis and, accordingly, some of the diabetic patients were never recognized, while (2) in contrast, other laboratory tests served as a check on the urinalysis, (3) the follow-up in the Rochon-Moran study was carried out by the family physicians who undoubtedly used different methods for further investigation and different criteria, while (4) in this survey, the follow-up work was done in one laboratory and the criteria remained constant.

The potential validity of the values obtained in this survey are heightened by some of the reports in the literature. Wilkerson and Krall[43] found 1.06 percent previously known diabetic patients in their Oxford study. Tabor and Frankhauser[41] observed a 1.09 percent prevalence in their group. Thus, these observations approach the 2 percent observation encountered in the 100 dental patients reported here.

Reason for Admission: Table 5 provides the unusual opportunity to observe the types of complaints with which diabetic as well as other individuals report when they first visit a dental clinic. It is apparent that one in two patients who report for routine care will prove to have a normal glucose tolerance pattern. It is very significant that of the three patients who reported with loose teeth, all of them showed evidence of decreased tolerance to carbohydrates. This is consistent with the reported literature.[12,13,15,16,19]

Family History of Diabetes Mellitus: Notwithstanding the reported genetic predictability of diabetes mellitus,[6] Table 6 shows that only one in four individuals found to be diabetic provided a positive familial history. It is especially interesting that approximately one in four of those individuals with a normal glucose tolerance pattern also reported a positive family diabetic history. Thus, apparently, one cannot rely upon this finding when one is dealing with the dental patient who is tentatively diagnosed as suffering with the oral manifestations of diabetes mellitus.

It is difficult to explain the discrepancy between the reported genetic predictability values and the findings in this survey. One point is clear. The percentage predictability[6] is derived from persons well-informed about their family tree. Most people are not well aware of the health of their relatives.

Consequently, this appears to be, at least, one important factor.

Symptoms: As has been previously reported, symptoms were divided into two categories: (1) *extraoral symptoms*, and (2) *intraoral symptoms*.

The *extraoral symptoms* studied were those frequently described as being associated with diabetes mellitus.

Boils: Though furunculosis is considered as a common finding of diabetes mellitus, Table 7 does not support this statement. This may possibly be due to the fact that: (1) boils do not appear early in the syndrome, and (2) in this investigation, the very earliest symptoms and signs of diabetes mellitus were elicited. This is underscored by the fact that, in the one individual who did report constant furunculosis, he proved to be suffering with diabetes mellitus.

Appetite: Apparently an increase in appetite is commonly associated with a decrease in sugar tolerance. Table 8 shows that one out of every four people in Group I complained with polyphagia and that this percentage was approximately twice that observed in the other groups. It is only fair to point out that the greatest reporting of decrease in appetite also appeared in the Group I patients. Perhaps, as in the case of furunculosis, polyphagia is not an early finding of carbohydrate imbalance.

Fluid Intake: Notwithstanding the frequent reporting that polydipsia is a common accompaniment of diabetes mellitus, this finding could not be substantiated as shown in Table 9. Again this might well be due to the fact that polydipsia is a symptom indicating more advanced diabetes and that, in this investigation, the more subtle stages of the disease were being detected.

Fluid Excretion: Increased fluid excretion (polyuria) occurred with greater frequency (Table 10) in Group I patients than in any other category. This is consistent with the general body of fact regarding the findings in diabetes mellitus.[26]

Weight Loss: The oft-mentioned weight loss as characteristic of diabetes appears to be supported as demonstrated in Table 11. The overwhelming percentage of such individuals were found in Group I and II.

The intraoral complaints previously reported in *Results* reveal rather interesting conclusions.

Dry Mouth: It appears from Table 12 that the greatest reporting of occasional dry mouth was noted in the Group I individuals. Of parenthetic interest is the fact that the three persons who reported *constant* dry mouth proved to be at the extremes of glucose tolerance with two showing decreased and one increased tolerance. It should be recalled that Sheppard[10] described xerostomia as constant or almost so in approximately 6 percent of his cases, occasionally present in 20 percent, and only rarely observed in 8 percent. We note from Table 12 that 8 percent of the diabetic individuals reported constant dry mouth while 46 percent indicated occasional xerostomia. The figures in this report are, therefore, quite consistent with the only other reported observations.

Burning Mouth: Sheppard[10] in his study of 100 diabetic patients made mention of the fact that 2 percent of his cases complained with a burning sensation in the oral cavity. Table 13 discloses that, in this report, 35 percent of the diabetic individuals described occasional burning mouth. The greater percentage of this finding in this study as compared to that of Sheppard is possibly due to the more elaborate laboratory testing utilized for this survey.

Gingival Tenderness: A number of investigators[11-17] have made mention of the fact that gingival tenderness is a common complaint among diabetic patients. However, no attempt has been made in these reports to quantitate this particular symptom. Table 14 shows that almost two out of every three Group I patients reported occasional to constant gingival tenderness in contrast to only one out of every three showing this same complaint in Group III

patients. Further discussion is unnecessary since the values obtained in this study cannot be compared to any others reported in the literature.

Pain with Tooth Percussion: A number of articles have appeared in the literature[12, 13, 15, 16, 18, 19] to indicate increased sensitivity of the teeth to percussion. However, no published quantitative studies are available. Table 15 shows that one out of four individuals in Group I reported slight to severe sensitivity to tooth percussion. This ratio is approximately twice that observed in any of the other categories. Parenthetic mention should be made of the fact that the one person who complained with severe sensitivity to tooth percussion was found to fit into Group I.

Dry Socket: The increased frequency of alveolar osteitis in diabetic patients has been mentioned without quantitative support by a number of investigators.[20, 21, 22] An examination of Table 16 appears to indicate that dry socket does not occur with greater frequency in diabetic patients. As a matter of fact, the evidence suggests somewhat that the converse may be true.

Signs: The signs previously reported in the section on *Results* are analyzed here in the same order.

Dryness of the Lips: According to Table 17, almost twice as many individuals in Group I demonstrated dryness of the lips than in any other category. Special mention should be made of the fact that the two persons who showed severe labial dryness proved to fit into Group I. This is consistent with the only report in the literature.[19] Possibly the mechanism for dryness of the lips is essentially that for xerostomia, and both are probably due to the generalized dehydration.

Gingival Stippling: It is difficult to draw any conclusions on the basis of slight loss of gingival stippling since, no doubt, many factors are operative. However, it is noteworthy that the four individuals in this survey who showed *severe* loss of gingival stippling all proved to fit into either Group I or II. Precisely why edema should be so extensive as to cause loss of stippling cannot be determined from this type of investigation. One group of investigators[23] has specifically indicated a 15 percent occurrence of spongy gingiva in their series of cases. It is difficult to draw any comparisons because of the categorization in Table 18 on the basis of severity. Perhaps, if one wishes to regard the "spongy gingiva" reported elsewhere as indicating moderate to severe loss of stippling, then the finding in Table 18 is consistent with the figures previously reported in the literature.

Gingival Bleeding: Actually, there is no such experience as spontaneous gingival bleeding. What is generally meant is that gingival bleeding occurs with relatively minimal trauma. It can be seen that bleeding with minimal trauma occurs much more frequently in Group I than in any other groups (Table 19). On the other hand, the commonly encountered gingival bleeding associated with relatively greater trauma (e.g. toothbrushing) is not the experience of the diabetic patient. The figure of 37 percent Group I patients with gingival bleeding is in line with the reported 32 percent[23] reported in the literature.

Gingival Hue: Minimal gingival alterations in color are not related to the glucose tolerance pattern. However, marked alterations in gingival hue appear to show some relationship (Table 20). This is borne out by the fact that blue to purple gingiva occurred four times as commonly in diabetic and prediabetic individuals than was observed in Group III with normal tolerance patterns. This finding is not in accord with the 92 percent previously reported.[23]

The high incidence (92 percent) was observed in a group of juvenile diabetic patients. It is known that diabetes mellitus in children is usually much more acute and severe. This may well explain, at least in part, the discrepancy.

Pocket Formation: It is noteworthy that the overwhelming percentage of individuals in Group I (91 percent) showed pocket

formation and that its occurrence was much greater in that group than in any of the others. This is consistent with the reported literature[23] which shows an incidence of 89 percent of diabetic children with periodontal pockets.

P. M. A. Index: Apparently, the overall prevalence of periodontal pathosis as measured by the P. M. A. index is much greater in the Group I patients than in the others. One encounters (Table 22) only one in three patients in Group I with relatively normal gingiva in contrast with almost one in two in Group III with normal glucose tolerance patterns.

Smooth Tongue: This sign has been reported in the dental literature as characteristic of diabetes mellitus.[10] However, no figures are available. According to Table 23, there is no evidence to indicate that smoothness of the dorsum of the tongue is a regular finding associated with disturbed carbohydrate metabolism.

Geographic Tongue: What has been said for smoothness of the tongue applies apparently also to geographic tongue as shown in Table 24.

Tongue Color: Redness of the tongue has been associated with diabetes mellitus. However, no figures are available in the literature.[10, 20] From Table 25 one can observe that one out of every five patients in Group I shows mild lingual erythema. Attention should be directed to the fact that mild redness occurred principally in those with extremes in sugar tolerance as shown by 19 and 15 percent in Groups I and IV.

Tongue Size: Though macroglossia has been suggested as a diabetic finding,[10, 20] evidence from Table 26 does not support the reported information.

Tongue Fissures: According to Table 27, tongue fissuring occurs much more commonly in individuals with normal glucose tolerance patterns and, therefore, does not support the evidence in the literature.[10, 18-20]

Tooth Mobility: Loosening of the teeth has been observed as a diabetic finding by a number of investigators[12, 13, 15, 16, 19] although no figures are given as to how frequently this observation can be made. According to Table 28, individuals in Group I showed the greatest occurrence of increased tooth mobility with almost three out of every four patients demonstrating this sign.

Number of Teeth: Tooth loss has been suggested as a diabetic finding.[14] Table 29 shows that patients in Group I had, on the average, approximately three teeth less than those in Group III with normal glucose tolerance patterns.

Since tooth loss increases with age in nondiabetic patients and since diabetes mellitus occurs with greater frequency in the older age groups, the question naturally arises as to whether tooth loss is truly a part of the diabetic syndrome. Table 34 outlines tooth loss in terms of age and carbohydrate status. It can be observed, in the main, that the patients with decreased glucose tolerance showed greater tooth loss. For example, after the age of 40, those in Group I had, on the average, four teeth less than those in Group III. This is fairly consistent with the literature reports.[26]

Calculus: It can be observed from Table 30 that approximately one out of every four patients in Group I showed no calculus whereas about four out of ten in Group III showed the same finding. Mention should be made of the fact that, from the standpoint of calculus, those individuals with increased glucose tolerance (Group IV) demonstrated the greatest absence of calculus.

Roentgenographic Findings: The two most discussed roentgenographic findings considered here are: (1) *alveolar bone loss*, and (2) *marginal periodontal widening*.

Alveolar Bone Loss: There has been general agreement in the literature that alveolar bone loss is part and parcel of the diabetic picture.[12, 15, 18, 19, 44, 45, 46] One investi-

gator[13] observed bone loss in 50 percent of a group of diabetic children. However, two groups[14,16] claim no alveolar bone loss. Table 31 shows that the normal bone pattern was not observed in any individual in Group I whereas it was encountered in one out of four with normal glucose tolerance patterns (Group III). One can observe from this chart that most of the diabetic patients showed considerable bone loss in contrast to the other groups.

Marginal Periodontal Widening: It has been reported in the literature that 70 percent of a group of diabetic children showed this roentgenographic finding.[13] Others have made the same observation.[16] One group has claimed that this finding does not occur.[23] From Table 32 one can observe that, in this report, 62 percent of those in Group I showed marginal to severe widening of the periodontal membrane in contrast to 39 percent of those in Group III. One can observe from Table 32 a decrease in the occurrence of this finding with an increase in sugar tolerance.

LABORATORY STUDIES

Table 33 shows that, had one relied exclusively upon urinalysis for the detection of diabetes, then nine patients in the entire series would have been suspected. As we have seen, twenty-six cases were found on the basis of the three-hour true glucose tolerance test. This means that the prevalence of detection increased three-fold by the use of more elaborate testing. This observation is quite consistent with the findings reported in other nondental epidemiologic studies.

SUMMARY AND CONCLUSIONS

One hundred routine dental patients were studied and information recorded regarding (1) general characteristics, (2) extraoral symptoms, elicited by a dental examiner, (3) intraoral symptoms, (4) intraoral signs, (5) periapical roentgenography, and (6) three-hour true glucose tolerance test. The following conclusions have been drawn:

1. A significant number of previously known diabetic patients (2 percent) appeared in the dental clinic for examination and care.

2. A highly significant number of patients (26 percent) who visited the dental clinic for examination and treatment showed evidence of *decreased* carbohydrate tolerance. Whether all or some of these individuals should be regarded as diabetic is basically a definition of the term diabetes mellitus.

3. Of those patients showing extremes of glucose tolerance (increased or decreased) the majority were of the female sex.

4. Every patient (3 percent) who reported to the clinic with a primary admission complaint of *loose teeth* also demonstrated *decreased* glucose tolerance.

5. A negative family history of diabetes mellitus proved of little help in eliminating the possibility of decreased glucose tolerance and possible diabetes mellitus.

6. In the main, *extraoral symptoms* (boils, appetite, fluid intake and excretion, and weight) did not contribute highly significant information regarding possible diabetes mellitus. Of the extraoral symptoms which were studied, fluid excretion appeared most diagnostic.

7. *Intraoral symptoms* proved quite helpful in the detection of possible diabetes mellitus. Dry and burning mouth, gingival tenderness, and pain with tooth percussion occurred more frequently in patients with decreased glucose tolerance.

8. Some *intraoral signs* (lip dryness, severe loss of gingival stippling, so-called spontaneous gingival bleeding, marked changes in gingival hue, absolute pocket formation, P. M. A. Index, tongue color, tooth mobility and loss, and presence of calculus) occurred with greater frequency in those patients showing evidence of decreased glucose tolerance.

9. The *roentgenographic findings* of al-

veolar bone loss and marginal widening of the periodontal membrane appeared more often in patients with confirmed and possible diabetes mellitus.

10. Because, in many instances, the findings in the patients with extremes of glucose tolerance (decreased or increased) were very similar, the relationship of blood glucose to oral findings irrespective of group categorization has also been studied. The results of that analysis will be reported in another publication.[47]

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GREATER MIAMI CHAPTER OF ALPHA OMEGA

Greater Miami Chapter of Alpha Omega announces its 2nd Annual "Sunshine Seminar" at the Deauville Hotel, Miami Beach, Florida, with sessions beginning January 22, 1960, and continuing through January 25. Clinicians for this year are Dr. Saul Schluger, Dr. Lewis Fox, Dr. Herbert Bartelstone and Dr. Leonard Hirschfeld. Theme of this year's seminar is "Periodontics for the General Practitioner." The tuition fee is $150.

For further information write Dr. Irving Gordon, 420 Lincoln Road, Miami Beach, Florida.

Guest Editorial . . . .

Periodontics Coming Into Its Own

In the 1930's a few dentists who confined their practice to scaling teeth were jokingly called root polishers. However, as specialists, they did a far better job in thoroughly removing subgingival calculus than the busy general practitioner. Even though most dentists realized that mouths treated by these specialists were in a more healthy condition, they believed the efforts of these specialists were hopeless and that the teeth would ultimately loosen and be lost. Hence many thought that subjecting patients to long hours of scaling procedures was an ordeal and a useless expenditure of money. The last thirty years, however, have brought proof that many teeth can be preserved for years of usefulness by periodontic treatment.

The original aim of dentistry was to treat and fill carious teeth and to replace lost ones. The keynote was repair and restoration. Placing small fillings so that large ones will be unnecessary and supplying missing teeth so as to maintain good occlusion and keep remaining teeth from drifting is not only reparative but also preventive.

The periodontist, on the other hand, practices prevention in a more realistic manner. Barring systemic complications, he is able with the complete cooperation of the patient not only to eliminate periodontal pockets but also to prevent their recurrence.

The fact that more teeth are lost through periodontal involvement than through caries is known generally; yet most seem indifferent regarding this sad state of affairs. Actually we are not indifferent, we are only resigned and take the hopeless attitude that "what can't be cured must be endured" There is, however, apparently no need for hopelessness, as the periodontist has shown that many lesions can be cured. Thus, we realize the great and urgent need for better and extended training in periodontology in dental schools.

To demonstrate the degree of growth of the specialty of periodontology, we may refer to a three-day workshop for teachers in periodontology, held at the University of California Medical Center last year, with 86 participants. At the workshop all phases of periodontology were discussed including the place this specialty has in the undergraduate curriculum. It is natural that any new subject (and periodontology is new in comparison to operative and prosthetic dentistry) has to fight for more teaching time. Periodontology, a truly preventive specialty, is indeed worthy of greater emphasis in the dental school curriculum.

Periodontology is a young specialty. Originally it was based entirely on the art of calculus removal with but little reference to the biologic sciences. Very soon, however, leaders in the specialty were not completely satisfied that periodontitis was due solely to the accumulation of calculus, and a systemic cause was sought. This phase, too, seems to have had a lively discussion in the workshop. On the question whether there is an established relationship between systemic disturbances and periodontal disease, a vote of 47 participants was divided.

All earnest leaders in periodontology realize that the future quality of service and its rating as an important specialty of dentistry is largely dependent on scientific research.

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If the service is purely symptomatic, viz. confined to removing constantly recurring calculus and reducing gingival inflammation, the specialty is merely an art dependent on digital dexterity. If, on the other hand, through research, a predisposing factor is defined which can be treated systematically, then the specialty takes on a new dignity and it becomes not only an art but also a science.

It is rather amazing that anyone should doubt the presence of a systemic factor as being related to periodontal inflammation. Nearly all diseases are influenced by systemic factors to a varying degree, and no one can deny that periodontitis is a disease. The fact that no systemic relationship has been defined up to the present is no proof that some systemic disturbance, as yet unknown, does not lower the resistance of the gingivae and make them prone to infection. It would be far better to open our minds and say, "we just don't know." *C. F. B.*

Book Reviews . . . .

The Management of Oral Disease—Joseph L. Bernier. *C. V. Mosby Company, St. Louis, 1959, 2nd Edition.*

The title of this book is slightly misleading because it indicates the primary purpose of the book is to discuss treatment. In keeping with the title, the format includes the heading "treatment" under the discussion of each disease, but the discussion of treatment is brief and in general very inadequate. This text is one of oral pathology and covers the field quite adequately and in sufficient detail to be of value to both the dental student and the practitioner. The text is well illustrated with both clinical and histological material and the illustrations are of better than average quality.

Some areas of the book are much stronger than others. The portion on odontogenic tumors, benign and malignant neoplasms is much stronger than the chapters on periodontal disease and dental caries.

The text contains many personal concepts of the author which are distinct variations from commonly accepted concepts and in some instances are more philosophical than scientific. However, they present the philosophy of the author which should be the accomplishment of a good text. The compilation of all the personal concepts expresses the author's philosophy of disease in general and of some diseases in particular. In this regard the text is unique and should make it of value to incite discussion in both graduate and undergraduate seminar type of teaching.

The five color plates have a high quality of reproduction and demonstrate well the features of the disease process they were selected to illustrate. The bibliography for some of the chapters is entirely adequate, but for others it is relatively brief and incomplete.

This second edition has eliminated many of the errors that are always inherent in a first edition. The book in general is provocative as well as instructive and should serve well its purpose to the profession and dental education. *D. K.*

Histogenesis of the Gingival Sulcus Epithelium in the Rat*

by GEORGE I. UOHARA, B.A., D.D.S., M.S.,** *Columbus, Ohio*

THE gingival sulcus is a circumferential groove which at its most apical border separates the clinical crown from the root. The gingival sulcus is of significant interest to many areas of dentistry.

A study of the formation of the normal sulcus should shed light on the sequence of normal events related to the emergence of the crown of the tooth into the oral cavity. In addition, it should describe the origin and formation of the epithelium of the sulcus.

This investigation, based on a chronologic, histologic study, describes the disposition of the elements of the enamel organ, its relationship to the oral mucosa prior to and during emergence and the origin and formation of the epithelial lining of the gingival sulcus.

This experimental study was accomplished on rat tissue and no implication is made that the same or identical events occur in the human. A comparison is made based on observations on human and other animal tissues made by other investigators.

REVIEW OF LITERATURE

Numerous observations on the eruption of human teeth and their associated structural changes have appeared in the literature. As early as 1771, John Hunter[1] wrote:

The teeth advance in their ossification and about the seventh, eighth, or ninth month after birth, the incisors begin to cut or pass through the gums, first, generally in the lower jaw.

The gum is not cut through by simple or mechanical pressure, but the irritation and consequent inflammation produces a thinning, or wasting of the gum at this point.

The earlier dental histology texts make sparse reference to the gingival sulcus and its origin.[2, 3, 4, 5] Based on human tissues, those of more recent date discuss the relationship of the formation of the sulcus to the enamel organ. They state that after the formation of the enamel rods, the ameloblasts produce a thin continuous layer, the primary cuticle, which covers the entire surface of the enamel. This cuticle is believed to be organically connected to the enamel matrix and becomes calcified. Upon completion of enamel development and maturation, the ameloblasts cease to be arranged in a well defined layer and can no longer be differentiated from the stratum intermedium and the outer enamel epithelium. These cell layers then form a stratified epithelial covering, the reduced enamel epithelium, which combines with the oral epithelium as the tooth approaches the oral cavity. During the emergence of the tooth, it is stated that the reduced enamel epithelium produces a keratinous secondary cuticle. The combined odontogenic and oral epithelium which covers the tip of the crown degenerates in the center and the crown emerges into the oral cavity. Upon emergence of the crown, the remaining reduced enamel epithelium then is known as the attached epithelial cuff and the groove that results from the separation of the epithelial cuff and the secondary cuticle on the enamel surface is the gingival sulcus.[6, 7, 8, 9, 10] These observations are based on the concept of epithelial attachment to enamel and limits the depth of the sulcus to the area of detachment of the epithelial cuff.[11, 12]

Another school of thought denies the presence of an epithelial attachment to enamel and states that when the tooth erupts, the capillary space between the tooth surface and epithelial cuff is the gingival sulcus which extends to the cemento-enamel junction.[13, 14] This older concept has been given renewed interest recently.[15]

Other investigators, while adhering to the concept of the epithelial attachment to enamel, have written that the formation and depth of the gingival sulcus is the result of a tear in the epithelial cuff rather than a result of a "peeling-out" process. They maintain that the bond between the cuticle and enamel is stronger than the union of the cells of the epithelial cuff.[16, 17]

It is also maintained that the normal sulcus is not formed between the secondary cuticle and the enamel epithelium or by an intra-epithelial split but between the surface of the oral epithelium and the more or less degenerating enamel epithelium. The fundus of

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the sulcus is found at the deepest area of separation of the oral and enamel epithelium. In a normal sulcus the ratio between the breaking down of the enamel epithelium and the simultaneous downgrowth of the mouth epithelium is constant.[18, 19] This view is further supported by the concept that the cells of the enamel organ do not change to keratinizing stratified squamous epithelium but that there is a successive replacement of the aging enamel epithelium by the oral epithelium.[20]

One of the most recent reports on the gingival sulcus states that the gingival epithelium migrates toward the cemento-enamel junction on the outer side of the reduced enamel epithelium which persists so that the epithelial cuff is of dual origin. The inner part is derived from the enamel epithelium and outer part from the gingival epithelium. The keratin layer of the gingival epithelium lies between the two and thereby forms the sulcus.[21]

METHODS AND MATERIALS

The Sherman strain of rat was used as the experimental animal. Litter mates of known ages are readily secured. The dentition is monophyodont and the molars are teeth of limited growth. They are similar to the human molar with the exception of the cusp tip which has no enamel.[22] The upper first molar was selected for study. In the rat, formation of the first molar begins at 13 days in utero and the tooth emerges into the oral cavity at 10 days after birth. This period of 28 days parallels the human first permanent molar of approximately six years.

Specimens were fixed at one day intervals from five to 25 days. Others were taken at 30 and 40 days and one adult at approximately 18 months of age. In all, 42 specimens were studied.

The tissues were decalcified, sectioned serially and stained either with hematoxylin and eosin for optical microscopy or acridine orange for fluorescent microscopy.

OBSERVATIONS

Serial sections of specimens of all ages reported in materials and methods were studied. Significant changes occurred at certain ages and only these will be described.

Five Day Specimens. The oral epithelium overlying the developing tooth was made up of a well defined keratin layer, a dark staining granular layer, a prickle cell layer and a basal layer with cuboidal cells and ovoid nuclei. The oral mucosa presented rete pegs except in the area of future emergence of the tooth.

The dental lamina, still intact, was continuous on its inner terminus with the outer tunic and its outer surface formed the surface of the oral mucosa in this area, where it presented a keratinized, granular and prickle cell layer continuous with those of the oral mucosa. The basal layer of the oral epithelium was continuous with and histologically identical to the basal layer of the dental lamina.

The outer tunic showed discontinuity in the occlusal area but was intact along the vertical walls to the cervix. The area of the stellate reticulum appeared to be overgrown, in part, by young connective tissue with capillaries in the area formerly occupied by epithelial cells. However, along the vertical walls of the tooth approaching the cervix, some portions of the stellate reticulum persisted at this period. Root formation had begun and Hertwig's epithelial sheath was evident.

Enamel formation had been in progress and the ameloblast layer consisted of tall columnar cells. The enamel was in a matrix state. The stratum intermedium was composed of squamous cells over most of the tooth but presented a cuboidal layer in contact with the ameloblasts (Fig. 1-A).

Nine Day Specimens. The developing tooth showed evidence of intra-cryptal eruption. The orifice of the bony crypt contained the lamina and adjacent submucosa. The inner surface of the crypt exhibited resorption on the occlusal portion indicating movement of the tooth toward the oral cavity.

The outer tunic had further lost its integrity on the occlusal surface and resembled epithelial rests within the submucosa which extended to the stratum intermedium. There was no change in the oral mucosa. There was no apparent alteration in the connective tissue of the submucosa or the dental sac. The number of cells and density of the fibers were similar to those observed at five days. The fibers of the oral submucosa were denser than those of the sac (Fig. 1-B).

On the lateral walls of the tooth, the four elements of the enamel organ, evident at seven days, presented the inner enamel epithelium, stratum intermedium, remnants of the stellate reticulum, and remnants of the necrobiotic outer tunic cells.

The stratum intermedium on the occlusal surface consisted of several layers of squamous cells with a single layer of cuboidal cells resting against the ameloblasts (Fig. 1-C). All the cells of the stratum intermedium were squamous at the cervix (Fig. 2-A).

The ameloblast layer consisted of tall columnar cells with well defined, dark staining, ovoid nuclei (Fig. 1C). Maturation of the enamel had begun at the tip of the cusp. The remainder was in the matrix state and amelogenesis had ceased. No histologic evidence of change was noted in the ameloblasts or cells of the stratum intermedium in areas of matrix and maturing enamel. Fluorescent microscopy showed a deeper orange color value for the ameloblasts at this time. The stratum intermedium and oral mucosa were yellowish blue.

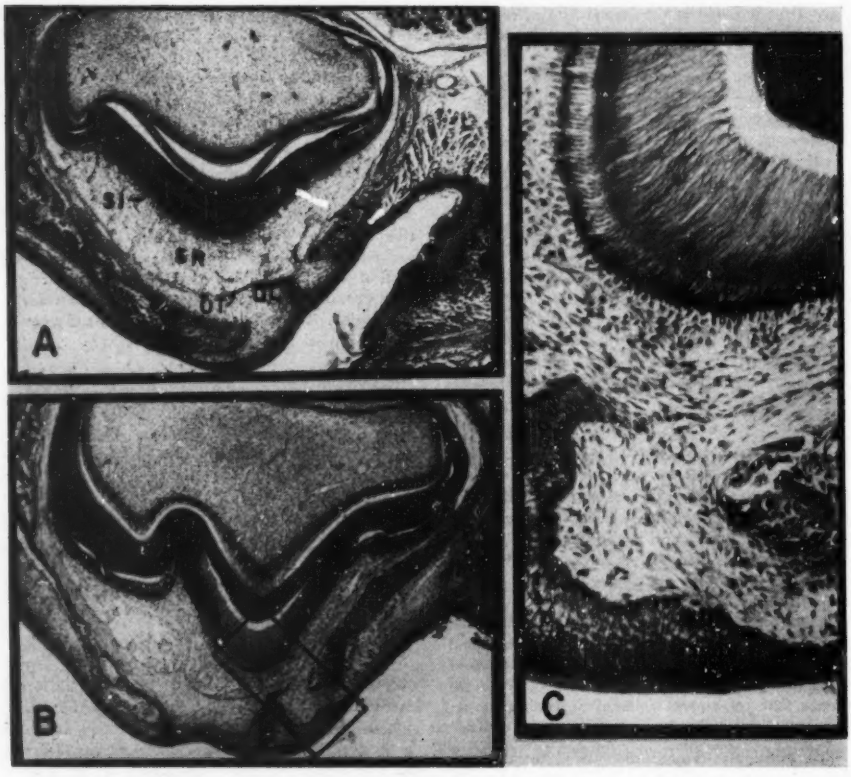

Figure 1

Eleven Day Specimens. The oral epithelium remained unchanged in appearance at this stage. The oral mucosa in the area of future tooth emergence exhibited no rete pegs. Lateral to this specialized oral mucosa the normal stippling of the mucosa and rete pegs were evident. The orifice of the bony crypt, enlarged by resorption, contained the dental lamina, epithelial rests of the outer tunic and submucosa (Fig. 2-B). Further intra-cryptal eruption established the first contact of the stratum intermedium with dental lamina and attached remnants of the outer tunic at the tip of the cusp (Fig. 2-B).

The cells of the stratum intermedium proliferated on the occlusal surface. The ameloblasts remained unchanged in all areas of the crown. At the same time, enamel maturation[23] to the stage of acid soluble enamel[24] had occurred at the cusp while a lesser state of maturation had thus far transpired along the lateral walls.

Twelve Day Specimens. The oral epithelium presented the same four well defined layers. The bony crypt enveloped the developing tooth to a lesser extent due to continued bone resorption widening its orifice.

Epithelial pearls, also observed in younger specimens, occurred in the lamina as well as in the remnants of the outer tunic. More cellular elements of the outer tunic and stratum intermedium were united at this time. On the vertical walls of the tooth, there was no trace of the outer tunic. Proliferation of the cells of the stratum intermedium had occurred following the pattern previously observed on the occlusal surface.

Fourteen Day Specimens. The oral mucosa remained unchanged. The tooth had moved toward the oral mucosa and bone resorption had advanced to further increase the orifice of the bony crypt. Enamel maturation was markedly advanced (Fig. 3-A).

More cells of the stratum intermedium were in contact with the lamina and outer tunic broadening the area of union. The underlying ameloblasts showed

no histologic change but the cuboidal layer of the cells of the stratum intermedium modulated to the squamous form (Fig. 3-B).

Sixteen Day Specimens. The lamina as a definite entity was no longer present due to its union with the stratum intermedium of the erupting tooth. Where union had occurred, the cells of the germinal layer of the oral mucosa and the stratum intermedium had lost their identity so that it was not possible to distinguish between them. Exclusive of the enamel organ there was no identifiable difference between these various joined epithelia either by ordinary histologic methods or fluorescent techniques.

Along the vertical walls of the tooth, the cells of the stratum intermedium had proliferated in a budding form. At the cervix the cells were squamous in appearance.

The orifice of the crypt had been resorbed to the greatest diameter of the tooth. Calcification was almost complete and the ameloblasts retained their columnar form over the entire crown of the tooth.

Seventeen Day Specimens. The orifice of the bony crypt had resorbed to permit emergence of the tooth. There was further movement of the tooth toward the oral cavity resulting in a wider union between the stratum intermedium and oral epithelium (Fig. 4-A). Along the lateral walls, the connective tissue of the tooth sac showed characteristics of a periodontal membrane, particularly at the cervix (Fig. 4-B). The ameloblasts covering the crown were in-

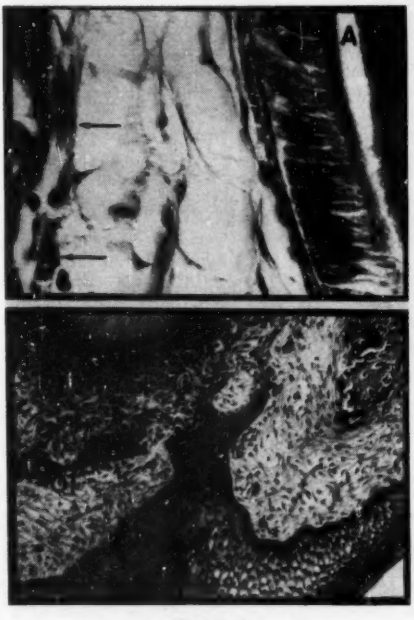

Figure 2

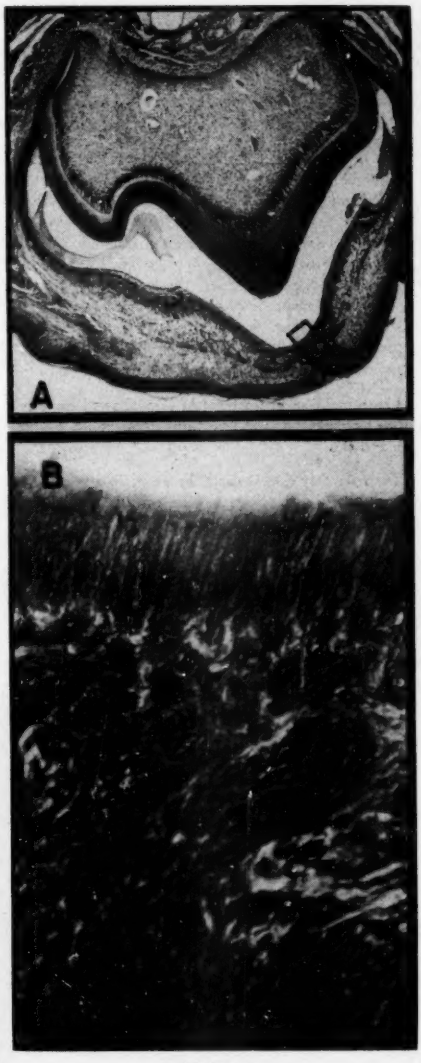

Figure 3

tact while the stratum intermedium showed budding proliferation along the vertical walls. The enamel was completely calcified except at the cervix (Fig. 4-A).

High power examination of the area of union of the stratum intermedium and oral epithelium revealed a layer of intact ameloblasts and a superficial granular layer. There was no nuclei at the surface indicating changes in the maturation of the epithelium in the absence of a basal layer (Fig. 4-C).

Eighteen Day Specimens. There was further occlusal movement of the tooth. The oral mucosa over the tip of the cusp had divided, leaving a single layer of columnar cells, the ameloblasts, as the sole covering of the cusp. There was no evidence of pressure causing the extrusion of the tooth through the gingiva (Fig. 5-A).

Under higher magnification (Fig. 5-B) the histologic changes in this area may be seen. The oral mucosa matured along the border of the opening exhibiting a normal keratinous surface which curved upon itself to form a cleft separating the oral mucosa from the ameloblasts. The ameloblasts covering the tip of the cusp were intact but showed early necrobiotic changes in the first exposed cells. The cleft between the oral mucosa and the ameloblast layer was the first suggestion of a gingival sulcus.

The remaining enamel epithelium showed no significant change from the previous illustrations (Figs. 4-A and B).

Nineteen Day Specimens. The cusp tip was visible macroscopically at this age. The orifice in the oral mucosa had increased in diameter. The ameloblasts covering the exposed cusp were no longer evident.

The cleft between the oral epithelium and the ameloblasts had extended farther apically (Fig. 6-A). Higher magnification of this area revealed degeneration of the ameloblasts between the enamel and the keratinized surface of the orifice (Fig. 6-B). The granular layer was apparent under the stratum corneum.

Proliferation of the stratum intermedium had increased along the vertical walls of the tooth diminishing in activity toward the cervix.

Twenty-one Day Specimens. Further emergence of the tooth into the oral cavity had occurred. The progressive changes in the oral mucosa associated with emergence followed the same pattern. The oral mucosa still covering the occlusal surface of the emerging tooth had no rete pegs. In contrast, the future mucosa of the outer papillary surface exhibited deep rete pegs (Fig. 7-A).

Budding epithelial proliferation of the stratum intermedium progressed cervically. The inner cells adjacent to the ameloblasts appeared squamous in the area near the cleft between the oral mucosa and ameloblasts while they retained their cuboidal shape at the cervical region.

The remaining ameloblast layer consisted of columnar cells with its long axis in the direction of the enamel rods. Fig. 7-B sheds light on the membrane concept of Nasmyth. The stratum corneum and granular layer invaginate to isolate the ameloblasts on the surface of the enamel. The ameloblasts show pyknotic changes in this area. The ameloblasts which remained adjacent to the exposed enamel resembled Nasmyth's membrane or may be interpreted to be the enamel cuticle.

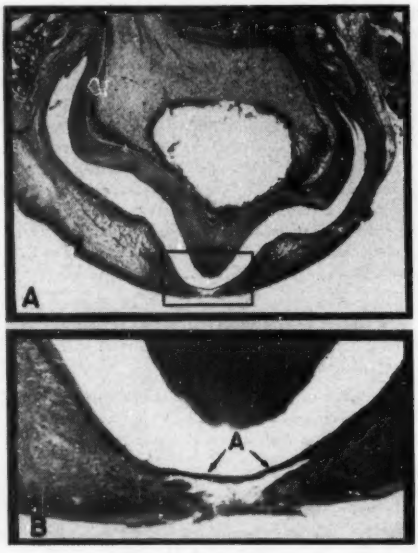

Figure 5

Twenty-two Day Specimens. After decalcification of the specimens and subsequent loss of enamel, it was impossible to establish histologically whether or not a sulcus existed. The origin of the epithelia lining the sulcus became evident at this time. The sulcus epithelia reflect two origins, one from oral mucosa and the other from odontogenic epithelium. The oral mucosa formed the marginal gingiva. It invaded the odontogenic epithelium undermining and isolating the ameloblasts. The oral mucosa, the portion of the sulcus epithelium toward the oral cavity, possessed a discrete basal layer showing normal maturation. The isolated ameloblasts were pyknotic and where separated by the oral mucosa presented a thin layer of degenerated cells. Further, apically the ameloblasts were columnar cells exhibiting a change in directional orientation. The directional relationship of the ameloblasts to enamel rods had changed to an acute angle inclining occasionally (Fig. 8-B).

Budding proliferation of the stratum intermedium had progressed to the cervix. The cells of the stratum intermedium adjacent to the ameloblasts had lost their cuboidal form and appeared squamous (Fig. 8-A).

Twenty-five Day Specimens. The tooth has fully erupted and, together with the surrounding soft tissues, established the normal adult relationships. The sulcus epithelium extended from the marginal gingiva to the cervix.

The two epithelia comprising the outer margin of the sulcus were still identifiable morphologically, by staining reaction and fluorescent microscopy. The

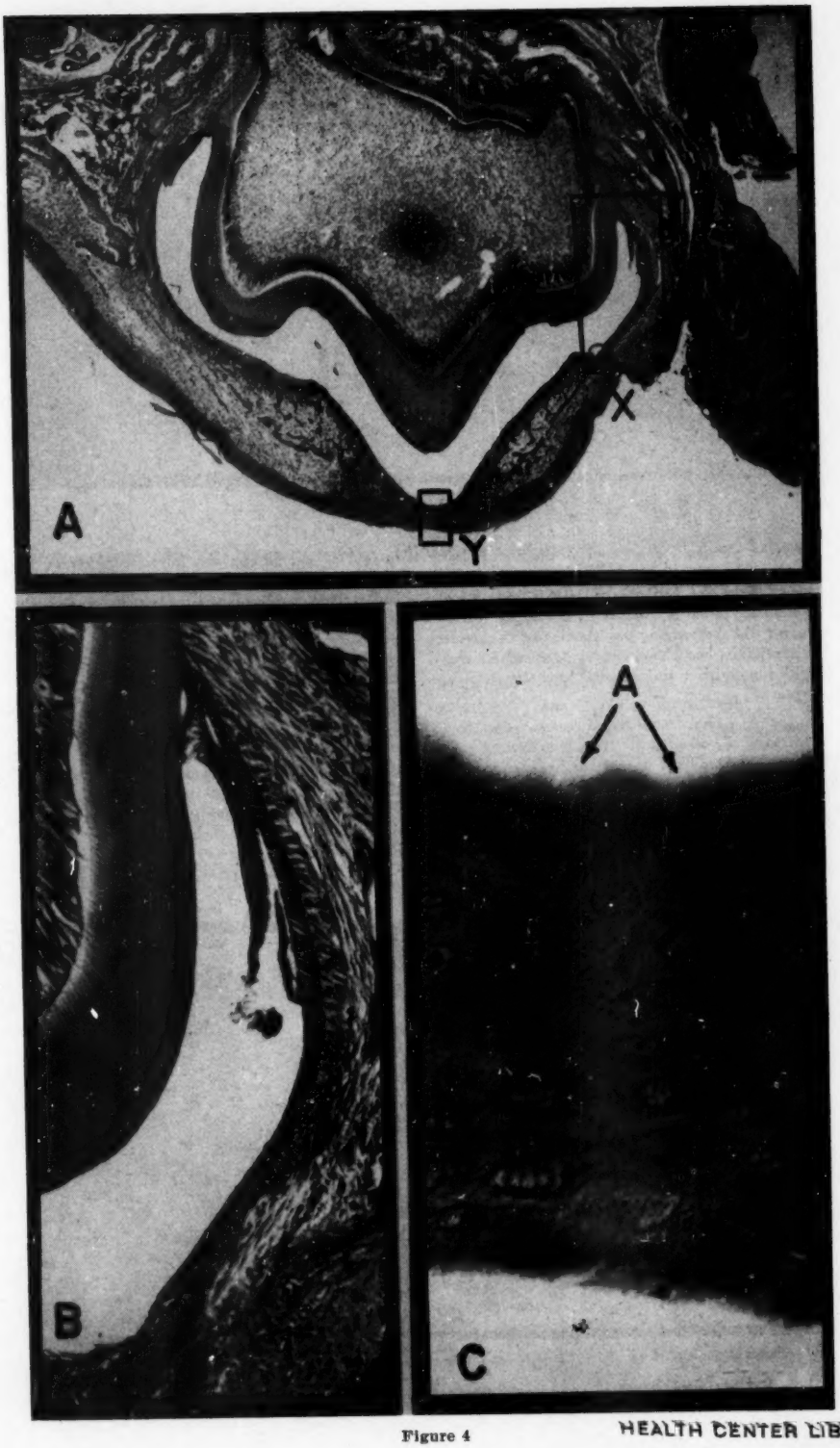

Figure 4

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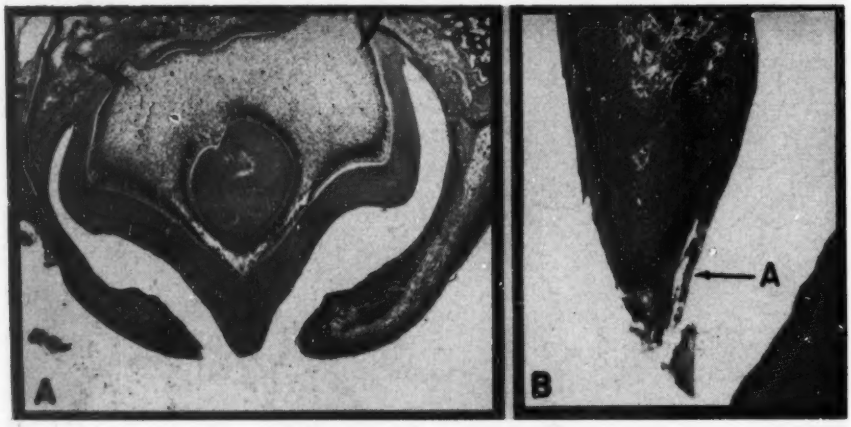

Figure 6

role of odontogenic and oral epithelium was clearly observed. The histologic appearance of the odontogenic epithelium had changed. Modulation of the ameloblasts from columnar to squamous had occurred and the budding proliferation of the stratum intermedium had changed to a layer of squamous epithelium with a distinct basal layer devoid of rete pegs.

The oral mucosa migrated toward the cervix separating the odontogenic epithelium from the submucosa. It descended behind the basal layer of the odontogenic epithelium (Fig. 9-A). At the fundus of the oral epithelium adjacent to the odontogenic epithelium there is a mitotic rich germinal zone. This area demonstrated round epithelial cells with no maturation toward the surface. Toward the margin of the gingiva the full maturation of the oral mucosa was evident. The normal granular layer and stratum corneum was evident underneath the necrobiotic odontogenic epithelium.

Further apically, the sulcus epithelium was of odontogenic origin. It was made up of squamous epithelium with a basal layer. There was relatively little mitosis and most of the cells were in the resting phase. No ameloblasts were present.

Fluorescent microscopy added to the interpretation of the sections. The oral mucosa portion of the sulcus epithelium was similar to the same tissue on the oral surface of the gingiva. The odontogenic epithelium presented two distinct portions. The occlusal area appeared granular, suggesting degenera-

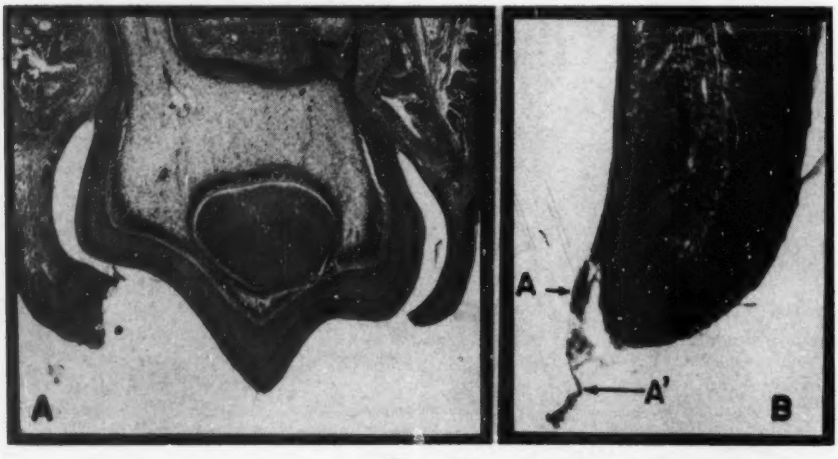

Figure 7

Figure 8

tive changes. In the fundus the cells appeared bright and homogeneous. The color of both the oral mucosa portion and cervical portion of the odontogenic epithelium was the same. The distinction between the two epithelia was made by the blue fluorescence of the stratum corneum.

Thirty and Forty Day Specimens. Of particular significance during this period was the presence of mitosis and resting cells in both the oral epithelium of the sulcus and the odontogenic epithelium.

The oral mucosa portion showed resting cells except at the zone of proliferation at its fundus. Similarly the odontogenic epithelium showed its major mitotic activity at the cervical area or its fundus. The newly formed cells appeared in the submucosa and their position indicates the process of apical migration. At no time was mitosis observed in the superficial cells of the odontogenic portion.

Eighteen Months. The sulcus epithelium had migrated to the surface of the cementum. The two origins of the sulcus epithelium, oral and odontogenic, were still evident histologically and by fluorescence. The oral mucosa retained the same anatomic relationship to the sulcus as did the odontogenic epithelium. In essence, the oral epithelium adjacent to the odontogenic epithelium resembled all other rete pegs of the oral mucosa in form but lacked basal cells toward the tooth where it presented a keratinized layer. The keratinizing layer of the oral mucosa portion separates it from the odontogenic epithelium. The odontogenic epithelium had proliferated apically along the cementum. The cells of the odontogenic layer were squamous. No stratum corneum or granular layers were evident (Fig. 9-B).

DISCUSSION

A developmental study was necessary to critically examine tissue changes and specifically the cytomorphosis of the cells in the area. In order to clarify the processes connected with the emergence of the tooth into the oral cavity it seems advisable to discuss the elements of the enamel organ and oral mucosa separately.

Oral Epithelium and Dental Lamina. The four layers of the oral epithelium were well defined in young rats. Rete pegs were absent in the oral mucosa overlying the crown but were present at the periphery of this area (Fig. 1-A and B). During the stage of intracryptal development, the oral epithelium was continuous with the persisting dental lamina (Fig. 1-C).

As the developing tooth approached the oral mucosa, budding proliferation of the stratum intermedium began to unite with the cells of the dental lamina (Fig. 2-B). Before the emergence of the crown into the oral cavity, changes in the oral epithelium were observed. The denal lamina and epithelial rests of the outer tunic, as definite structures, were not discernible due to their union with the cells of the stratum intermedium (Fig. 4-A). In the area of

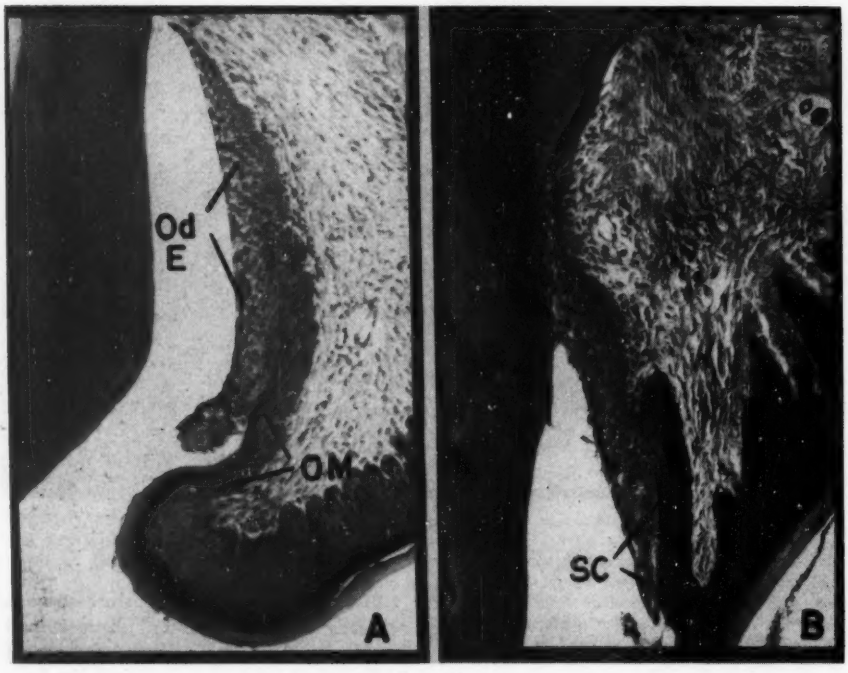

Figure 9

union, the oral epithelium was thin and identification of the various cellular layers was not possible. The combined oral mucosa and odontogenic epithelium over the cusp presented several layers of epithelial cells with keratohyalin granules resting on a single layer of ameloblasts (Fig. 4-C).

At the time of emergence of the crown, the oral epithelium spontaneously opens over the tip of the cusp. The stratum corneum turned upon itself, leaving the ameloblasts as the sole covering of the enamel in such a way that the ameloblast layer remained intact and continuous (Fig. 5-B).

As further emergence took place, the oral epithelium migrated behind the ameloblast layer (Fig. 6-B). Ameloblasts over the tip of the cusp were no longer evident and those isolated by the oral mucosa showed degenerative changes (Fig. 7-B). The cuboidal cells of the stratum intermedium were transformed into squamous epithelial cells. This transformation began near the ingrowth of the oral epithelium and progressed cervically. Subsequently, the ameloblasts in this area modulated to form squamous cells (Fig. 8-B).

Mature specimens revealed that the sulcus epithelium was composed of oral epithelium at the occlusal half.

Bony Crypt. At nine days, the developing tooth was still enclosed in a well developed bony crypt except for the orifice which contained the lamina (Fig. 1-B). Resorption was observed on the tooth side of the crypt which continued until a few days before eruption, enlarging the orifice to accommodate the periphery of the tooth (Fig. 5-A). It is interesting to note that epithelial changes in the oral mucosa occurred before the orifice of the crypt was large enough for the tooth to emerge.

At the time of tooth emergence, the bone was at a level occlusal to the cemento-

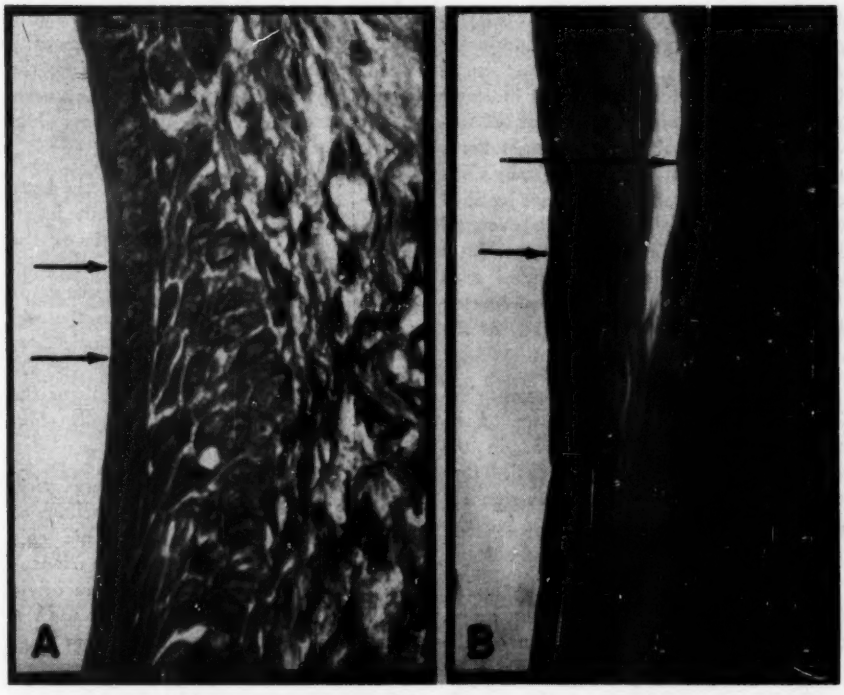

Figure 10

enamel junction; at 25 days it was at the junction and at 30 days it was apical to the junction. In the 18 month specimen, where there had been epithelial migration along the surface of the cementum, the vertical distance between the cemento-enamel junction and height of the bone had increased (Figs. 1-C and 8-A).

Outer Tunic. Generally, it has been stated that in the human dentition, the outer tunic participates in the formation of the reduced enamel epithelium.[6, 7, 9] This was not observed in this study.

At nine days post partum, the outer tunic was observed as epithelial rests, especially in the occlusal area. Fibroblasts and capillaries penetrated the outer tunic (Fig. 1-B). Lefkowitz, Bodecker and Mardfin have stated that capillaries and fibroblasts from the follicular wall penetrated the outer tunic, leaving a residue of epithelial rests.[25] Others have reported similar findings.[26, 27] Along the vertical walls, the outer tunic began to degenerate (Fig. 2-A) and at 11 days it was no longer evident.

Subsequently, the outer tunic remnants associated with the lamina on the occlusal aspect combined with the proliferating cells of the stratum intermedium as the tooth moved toward the oral cavity. This process continued in the cuspal area where union of the lamina and the stratum intermedium took place (Fig. 3-A). Identification of the cellular layers in the united epithelia was not possible except for the ameloblasts (Fig. 4-C).

Epithelial pearls of dyskeratosis were seen in many specimens in the oral mucosa, dental lamina and remnants of the outer tunic. These occurrences indicated the maturation potential of the cells of the oral mucosa, dental lamina and outer tunic. No keratinization was observed in the stratum intermedium and ameloblast layers. This

finding indicates that the latter two layers of the enamel organ differ in specificity from the cells of the oral mucosa, dental lamina and outer tunic. It further indicates that the odontogenic epithelium of the sulcus may be incapable of forming a keratinous cuticle as it descends apically. The designation of outer tunic appears justified because the potential of these cells is toward keratin formation and not enamel. It is possible that some of the epithelial rests found in human dental follicles as reported by Conklin and Stafne[28] were rests of the outer tunic.

Stellate Reticulum. Many studies relating to the function and disposition of the stellate reticulum have appeared in the literature.[7, 8, 29, 30] Some have revealed that the cells of the stellate reticulum were replaced by capillaries and connective tissue.[25, 27, 31, 32]

At the time when most of the developing tooth was still enclosed in the bony crypt, the area of the stellate reticulum on the occlusal had been replaced, in part, by capillaries and fibroblasts. Traces of the stellate reticulum were seen along the vertical walls of the tooth at the cervix. At the time of resorption of the bony crypt, the remaining portion of the stellate reticulum at the cervix had been replaced by connective tissue (Figs. 1-B, 3-A).

Stratum Intermedium. Numerous reports concerning the stratum intermedium reflect differences of opinion among the investigators.[7, 8, 9, 33] At birth, the inner cells of the stratum intermedium were cuboidal in the occlusal area while they remained squamous at the cervix.[25] Early post partum specimens showed proliferation of the stratum intermedium at the occlusal surface whereas at the cervix they presented only two layers of squamous cells. Variations in the number of cell layers may be the result of oblique sectioning.

When the bony crypt had been resorbed to permit the tooth to emerge, the stratum intermedium united with the dental lamina on the occlusal. Along the vertical walls, the stratum intermedium showed budding proliferation which proceeded cervically until the emergence of the tooth into the oral cavity. The cells adjacent to the ameloblasts remained cuboidal at this time (Fig. 4-A).

After emergence of the tooth, the layer of cuboidal cells of the stratum intermedium along the vertical walls of the tooth transformed to squamous cells (Fig. 8-B). This transformation took place gradually and continued cervically as the oral epithelium migrated behind the ameloblasts in the occlusal area. Soon thereafter the ameloblasts along the vertical walls of the tooth modulated to squamous cells (Fig. 9-A).

The sulcus epithelium of mature specimens was composed of oral epithelium at the occlusal half and odontogenic epithelium at the cervical half. The odontogenic half of the sulcus epithelium originating from modulated ameloblasts and the stratum intermedium had no rete pegs (Fig. 9-A). While in a resting state, the cells of the odontogenic epithelium adjacent to the connective tissue presented the appearance of basal cells.

Older specimens showed a similar relationship with oral epithelium situated occlusal to and behind the odontogenic epithelium. However, there had been a migration of odontogenic epithelium along the surface of the centum. The newly divided cells were found as isolated epithelial cells in the submucosa just beyond the original mass. The migrating cells appeared squamous, and no basement membrane was seen at its deepest portion nor was degeneration of the connective tissue obvious. Investigation through the use of a reticular and other stains may provide some information on the mechanism of epithelial proliferation on the cementum.

Ameloblasts. During enamel formation, the ameloblasts were tall columnar cells with well defined ovoid nuclei. They were bounded on one end by the enamel matrix and on the other by the cuboidal cells of the

stratum intermedium. The cytomorphosis of the ameloblasts has been carefully studied by many investigators[11, 20, 23, 34, 35, 36] in human and animal specimens.

When the tooth began to emerge into the oral cavity, the superimposed oral epithelium opened and the cusp, covered with a layer of ameloblasts, was exposed to the oral cavity. The ameloblast layer maintained its integrity and continuity to the rest of the ameloblast layer, and was in contact with enamel (Fig. 5-B). Later, that portion of the ameloblast layer exposed to the oral cavity and undermined by the oral epithelium showed degenerative changes (Fig. 6-B).

The ameloblasts along the vertical walls modulated to squamous cells and together with cells of the stratum intermedium formed part of the sulcus epithelium (Figs. 8-B, 9-A). Histological evidence cannot explain this change. Similar findings have been reported by Ussing.[37]

Reduced Enamel Epithelium. The concept of the reduced enamel epithelium in the human is defined by the statement: "The ameloblasts shorten after the enamel cuticle is formed, and the epithelial cells comprising the enamel organ are reduced to a few layers of cuboidal cells which are then called reduced enamel epithelium."[38]

Changes in the epithelial layers of the enamel organ of the rat follow a different course. In a pre-natal odontogenesis study of the rat molar, the replacement of the stellate reticulum by young connective tissue over the crown of the tooth was described.[25] The separation of the outer tunic from the stratum intermedium and ameloblastic layer was completed by the connective tissue replacement of the stellate reticulum in two ways. On the occlusal surface of the tooth the outer tunic is separated from the stratum intermedium and the ameloblasts (Figs. 1-A, B). Intracryptal eruption caused them to join again to form a single layer just prior to emergence. The stratum intermedium on the occlusal surface united with the outer tunic, dental lamina, and the oral mucosa and participated in the mucosal opening which permitted the tooth to emerge.

On the vertical walls, the area of the stellate reticulum was occupied by connective tissue and the outer tunic degenerated before maturation of the enamel occurred. At this stage the ameloblasts and stratum intermedium had lost their connection with the outer tunic. The odontogenic epithelium which originated from the ameloblasts and stratum intermedium persisted and proliferated apically.

Enamel Cuticle. In the human dentition, the primary cuticle is believed to be a product of the ameloblasts and is largely organic. The secondary cuticle is described as a keratinous layer deposited on the surface of the primary cuticle by the reduced enamel epithelium.[38]

No evidence was found in this study to indicate that the primary cuticle was present. Figure 10-A illustrates what is commonly described as the primary cuticle. Critical examination of this area indicated that the presumed cuticle may be an optical phenomenon caused by the thickness of the section and not a separate anatomic entity. The same optical result was observed on the surface of the stratum corneum which was a keratinized layer (Fig. 10-B). On the basis of this interpretation, existence of a largely organic primary cuticle on the rat molar is questioned.

The secondary cuticle on the occlusal surface could be formed by the ameloblasts only (Fig. 5-B). Critical examination of the surface revealed that the cuticle was a layer of degenerated ameloblasts (Fig. 7-B). Along the vertical walls, the odontogenic epithelium composed of stratum intermedium and ameloblasts, either as columnar or as their squamous derivatives, degenerated to form a surface layer when isolated by the ingrowth of the oral mucosa.

Emergence. The emergence of the tooth into the oral cavity is a dynamic process in which all associated parts play their indi-

vidual roles. There was a harmonious interplay of processes between the emerging tooth, the bony crypt and the oral mucosa. It has been expressed that during the emergence of a tooth in the human, the connective tissue is compressed and the tooth caused bulging and blanching of the overlying gingival tissue.[39] The animal specimens studied in this experiment did not show these changes. No histologic changes were observed in the submucosa (Figs. 4-B, 5-A).

The oral mucosa in the area of future emergence presented a specialized type of stratified squamous epithelium devoid of rete pegs. This area remained relatively inactive until the union of the stratum intermedium and the overlying epithelial layers. Degeneration of the oral mucosa was spontaneous and occurred before the tooth emerged. In no case was the oral mucosa seen stretched over the emerging tip of the cusp, nor was there any elevation of the oral mucosa. On the basis of this examination, the oral mucosa in the area of the emerging tooth appeared to be endowed with the function of dividing in harmony with eruption. The separation of the oral mucosa occurred just prior to the tooth reaching the level of the outer layer of the oral mucosa. It is equally interesting to note that no evidence of inflammation was observed during the process of emergence.

The eruption of the tooth is associated with resorption of the submucosa. Engel, in an interesting study, reported chemical changes in the ground substance of the connective tissue associated with eruption.[40] Orban[38] states: "It is probable that the epithelial cells elaborate an enzyme that is able to destroy connective tissue by eesmolysis." The epithelial separation which occurred over the tip of the cusp in the absence of connective tissue may be due to specificity on the part of these cells.

Apical Migration of Odontogenic Epithelium. The odontogenic epithelium, possessing a growth pattern of bud-like proliferation[41] assumed the same pattern in its apical migration. The layer of cells resting against the enamel or cementum did not mature to keratin. In contact with a calcified surface it is inconceivable that they proliferate toward the surface. In the absence of inflammation[42] these cells remained in a resting state. A proliferative zone at the fundus was observed. Newly divided young epithelial cells were found in the periodontal membrane just outside the mass of epithelium. No basement membrane was observed in this area at the time active division took place. Examination of all sections showed periods of rest and proliferation.

Epithelial Attachment. This study was made with calcified sections in which the enamel was either observed in the matrix state or in various stages of maturation. It was not possible to establish an epithelial attachment to enamel from the time of maturation since the enamel was dissolved during decalcification. Attachment is a physical phenomenon better expressed by the term "bonding," which is a joining of two substances. "Bonding" is tested or proved by physical methods. The presence or absence of an epithelial attachment to enamel can best be determined clinically[15] and any effort to establish an epithelial attachment to enamel with decalcified sections is perplexing. Not until the odontogenic epithelium migrates apically along the surface of cementum can any statement be made on the basis of histological examination (Fig. 9-B). The cementum, unlike enamel, is retained after decalcification and either the epithelium in contact with it or the sulcus between the epithelium and cementum can be observed (Fig. 9-B). The sulcus when observed in histological section is bounded on the tooth side by cementum and enamel. The odontogenic epithelium is in contact with the cementum further apically and the sulcus is the space between the epithelium and hard tissue. There was no histologic evidence to establish that the epithelium of the sulcus was attached to the enamel. An intra-epithelial split[17] was not in evidence.

CONCLUSIONS

1. The opening of the mucosa which permits emergence of the tooth is a spontaneous process.

2. The ameloblasts on the occlusal surface persist until emergence of the tooth in the case of the rat molar.

3. Neither the primary or secondary cuticles were established on a histologic basis.

4. The epithelium of the gingival sulcus arises from the oral mucosa and odontogenic epithelium.

5. The outer tunic and stellate reticulum do not contribute to the formation of the sulcus epithelium.

6. The odontogenic epithelium is derived from the stratum intermedium and ameloblasts which arose from the vertical wall of the tooth.

7. The oral mucosa forms the marginal gingival and extends apically behind the occlusal part of the odontogenic epithelium.

8. The odontogenic epithelium forms the fundal half of the sulcus epithelium.

9. Apical migration occurs at growth centers in the fundi of both odontogenic and oral epithelia.

10. Epithelial attachment to enamel cannot be established by histological sections.

11. On the basis of maturation, the outer tunic, dental lamina and oral mucosa are related.

12. The apical limit of the gingival sulcus can be established histologically only when a separation of the epithelium and cementum is evident.

ACKNOWLEDGMENT

The author wishes to express his deep appreciation and indebtedness to Dr. William Lefkowitz, Professor of Dentistry, Ohio State University, for his untiring efforts and guidance throughout the course of this study. Sincere appreciation is also acknowledged to Dr. John Wilson, Chairman, Department of Periodontics, Ohio State University, for his advice and encouragement.

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FORT WORTH DISTRICT DENTAL SOCIETY PROGRAM FOR 1959-60

Dr. Bert L. Means, President of the Fort Worth District Dental Society, announces that the following programs have been arranged for this year:

Tuesday, November 10, 1959, Emotional Significance in Dental Practice, Dr. Nathan Friedman; Occlusion, the Restorative Problem and the Periodontium, Dr. Saul Schluger.

Tuesday, January 12, 1960, The Nature and Significance of Mucosal Keratotic Lesions, Dr. Edward V. Zegarelli.

Tuesday, February 9, 1960, Problems of Occlusion, the Rationals for their Correction, Dr. Robert F. Moyers.

Tuesday, March 8, 1960, Surgical and Prosthetic Management of Advanced Periodontal Disease, Dr. John F. Prichard.

For further information write Dr. Travis L. Lanham, Program Chairman, Fort Worth District Dental Society, 2928 West Sixth Street, Fort Worth, Texas.

The Use of Modified Rubber Base Material as a Radiopaque Substance in Periodontal Roentgenography

by ALBERT SALKIND, D.D.S.,* AND SOL. J. EWEN, D.D.S.,** *New York, New York*

THE development of a satisfactory radiopaque substance for periodontal roentgenography could be helpful in the diagnosis and treatment of periodontal disease.

There are many diagnostic aids used in periodontics for proper evalution of the depth and topography of periodontal pockets. These are the periodontal probe, gutta percha points, Hirschfeld points, solutions, suspensions, and semi-solids to evaluate the depth, extent, and configuration of periodontal defects. Of these, the radiopaque semi-solids[1,2] offer promise of revealing most accurately the various contours of periodontal deformities.

Clinically, the periodontal probe is useful in determining pocket depth. Yet, in order to gain complete access, it may become necessary to angle the probe, offsetting the possibility of an accurate millimeter reading. Roentgenographically, gutta percha and metal points reveal the pocket depth and gingival height accurately. However, they neither demonstrate the individual contours, nor the lateral configuration of any given periodontal pocket.

Heretofore, the radiopaque solutions, suspensions, and semi-solids, have had limited use. The methods of manipulation, the inadequate opacity, the poor flow control, have served to make radiopaque materials difficult to use. In addition, shallow periodontal pockets are unable to receive an adequate amount of the material; calculus and granulomatous tissue tend to hinder the flow of radiopaque substance, and the bulkiness of this substance may distort the configuration of the lesion.

In an historical review of the uses of radiopaque substances in general dental roentgenography, A. Porter Sweet[3] mentions the use of gutta percha and lead sulphate in 1897 as contrast media. In 1904 barium sulphate was used. In 1916 thorium oxide, sodium iodide, and sodium bromide were utilized. In 1940, complex salts and organic substances came into vogue. Uroselectan, sodium-tetra-iodo-phenolphthalein were a few among many studied and developed. In periodontal diagnosis, Goldman[4] describes the use of lipiodol or a paste of bismuth subnitrate and glycerin inserted into the periodontal pockets. Richards[5] has listed carbon tetrachloride, sodium iodide in solutions varying from 20% to 80%, non-toxic pastes of barium sulphate and calcium carbonate, and a mixed paste of sodium iodide and bentonite. As indicated above, these substances have all been inadequate, and have not been accepted in daily practise to aid in the diagnosis and treatment of periodontal pockets.

For a radiopaque solution, suspension, or semi-solid to be acceptable as a contrast medium in periodontal roentgenography, it should have the following qualifications:

1. Ease of introduction between the gingiva and the roots of teeth.

2. Ease of penetration to the full depth and topographical outline of the pocket.

3. Ability to produce an opacity clearly discernible from the immediately surrounding hard tissues.

4. Non-irritability to the already inflamed tissues.[6]

5. Readily removable.

For the purpose of achieving these quali-

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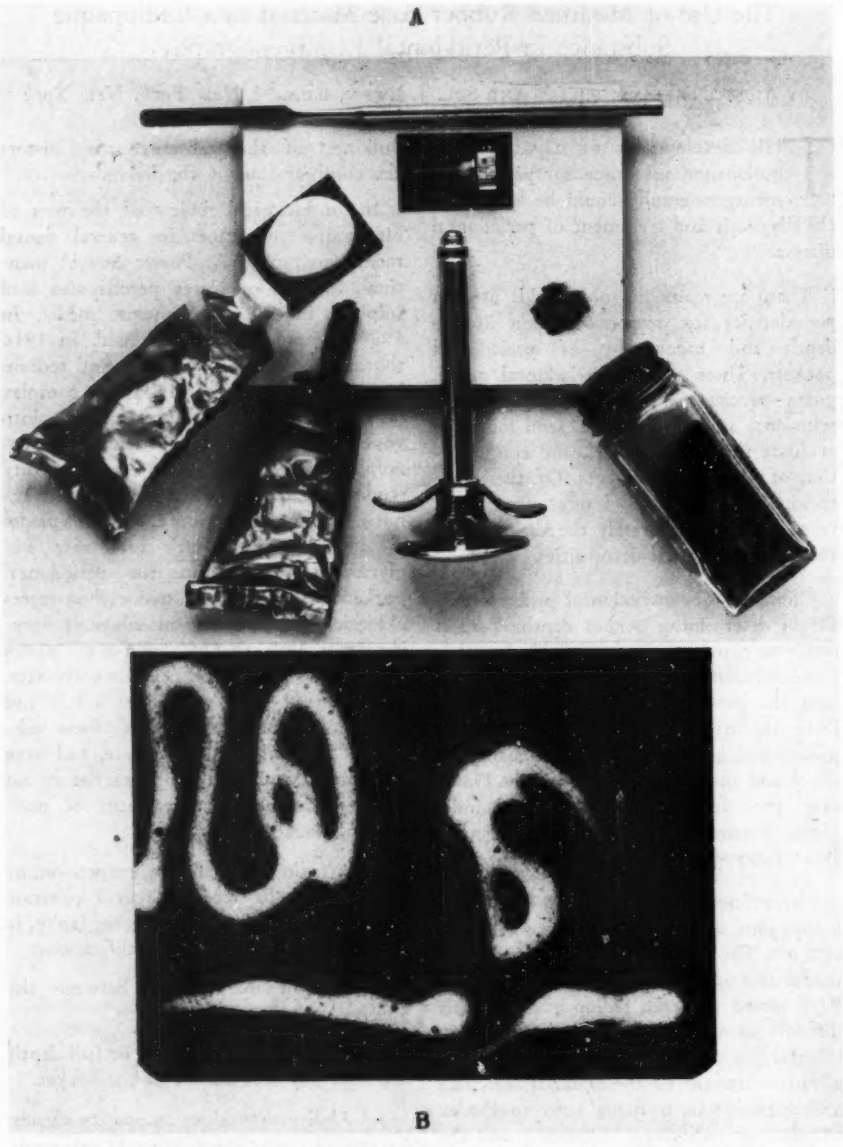

Fig. 1. A. Tubes of rubber base material, aluminum gun, silver alloy, mixing pad, and spatula. Note newly designed plastic tip (center). B. Roentgenograms of expressed radiopaque rubber base material.

fications, a study was made, employing a material and method to illuminate the topography and character of periodontal defects in roentgenograms.

MATERIALS AND METHODS

The following materials were used in this investigation·

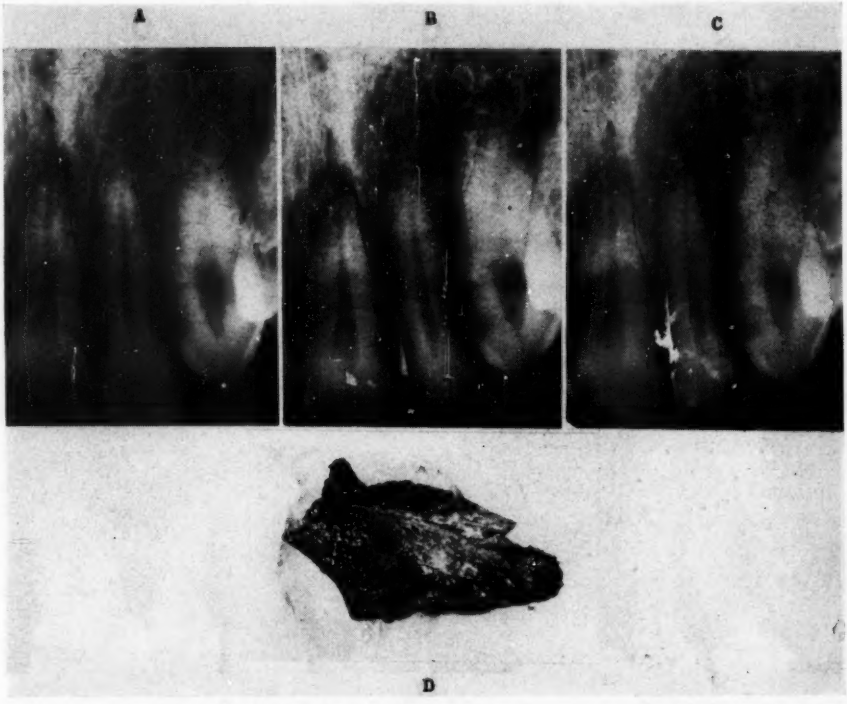

Fig. 2. A. Upper left lateral. B. Same area with gutta point in place. C. Same area with radiopaque substance in place. Beaded portion indicates location of gingival margin. D. Three dimensional rubber base alloy duplicate of mesial pocket in toto.

1. Kerr's light bodied permlastic rubber base impression material.

2. Finely ground silver-amalgam alloy.

3. Aluminum gun with a newly designed ultra-fine lumen plastic tip.

4. Vaseline.

5. Cotton points and college pliers.

6. Mixing pad.

The lumen of the plastic tip and the interior of the aluminum syringe are lubricated with vaseline. Equal parts of rubber base, accelerator and alloy are placed on a mixing pad. (Fig. 1A). The alloy is first mixed into the white rubber base until an even gray color is obtained. The area to be x-rayed is isolated with cotton rolls, and the pocket dried with warm air and cotton points which are retained in situ until pocket is to be filled with radiopaque material. At this time, the rubber base-alloy mix is combined with accelerator and after spatulation into a smooth paste, is fed into the plastic tip, and the barrel of the syringe. Pressure is then applied to the plunger to express whatever vaseline lies within the gun and tip. Once some of the rubber base-alloy has been expressed (Fig. 1B), it is ready to be placed within the periodontal pocket. Cotton points are removed from the pocket, and the tip placed to the depth of the pocket, and withdrawn slightly. The medium is then expressed from the gun until it appears above the gingival margin. In this fashion, the material is injected into the pockets from the buccal and lingual approaches. Excess is carefully wiped off level with

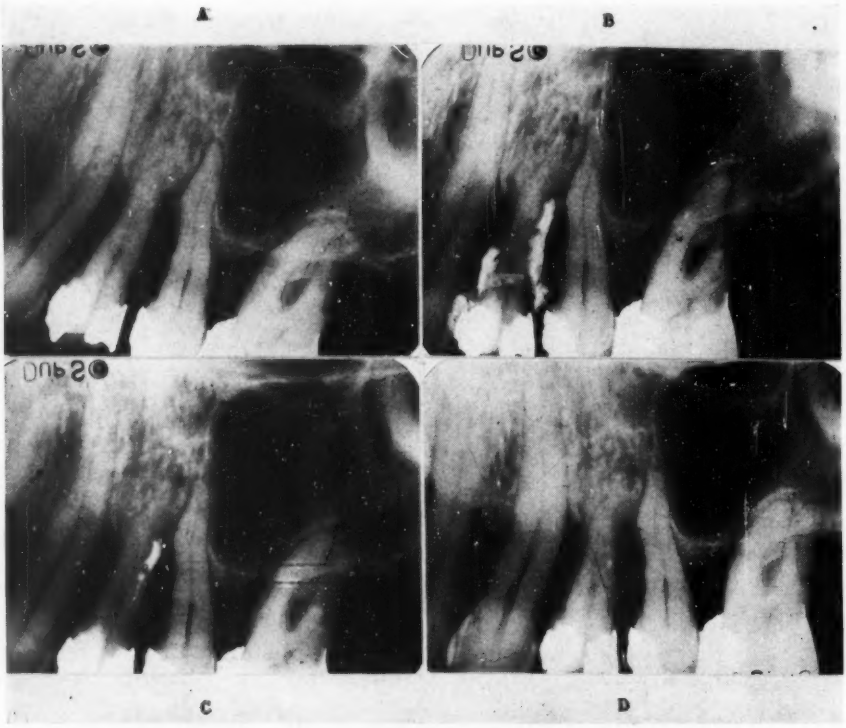

Fig. 3. A. Upper left first bicuspid area. B. Same area with radiopaque material in place. Note a bridge of radiopaque material within buccal gingival sulcus, connecting mesial and distal pockets. C. Remnant left in pocket. D. Same area with material removed.

the gingival margins. Roentgenograms are taken immediately. Shortly thereafter, when the mix has attained its usual semi-rigid firmness, it is carefully lifted out of the pocket with an explorer or college plier. In many instances, a three dimensional replica of the pocket is made available.

In this investigation, roentgenograms were taken of each patient beforehand. The material was then placed within the pocket, and a second radiograph taken. Once the material had set, it was removed from the pocket, and a third film taken, to determine whether the material had been completely removed.

OBSERVATIONS

For purposes of demonstration in this report, four patients ranging from ages 36 to 49 years, have been selected at random from the many upon whom this method has been tried.

In a patient with moderate periodontitis, an upper left lateral incisor was selected for radiographic study (Fig. 2A). A gutta percha point was inserted to the base of the pocket and cut at the gingival margin (Fig. 2B). After the point was removed, the radiopaque material was prepared, and inserted into the pocket following the procedure outlined previously. A roentgenogram of the area is shown in Fig. 2C. When the material had set, it was removed in toto with an explorer. This rubber form was a three dimensional duplicate of the pocket (Fig. 2D).

Figs. 2A, B, C, D, show that a semi-

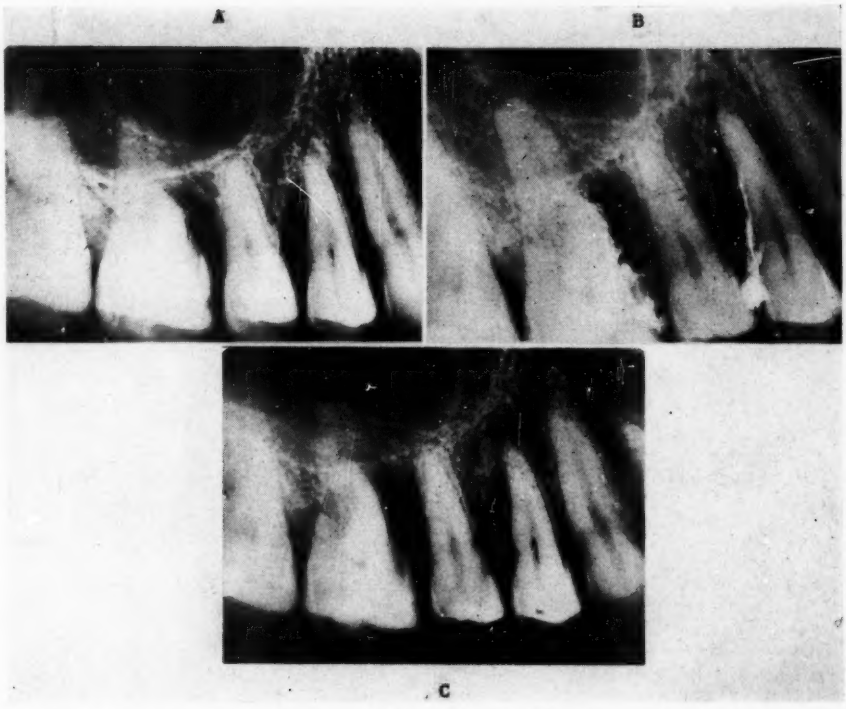

Fig. 4. A. Upper right molar-bicuspid region. B. Same area with radiopaque material in place. Note that granulation tissue confines radiopaque material to a capillary-like space. C. Same area with material removed.

solid rubber base-alloy contrast medium penetrates to the very base of the pocket. The medium particularly reveals the minute amount of space which lies between the soft tissue wall and the hard tissue side of the pocket.

In a patient with advanced periodontitis, the upper left first bicuspid was selected for radiographic study (Fig. 3A). Mesial and distal pockets were dried with cotton points and warm air. The material was placed within the pocket until it appeared above the gingival margin. (Fig. 3B). When the material had hardened, it was removed with an explorer and a new radiograph was taken. Some of the radiopaque material remained behind (Fig. 3C). This was removed. Fig. 3D indicates the absence of any remaining radiopaque material. Fig. 4 illustrates infrabony pockets in a patient with advanced periodontitis. Fig. 4A—the infrabony pocket. Fig. 4B shows the radiopaque material after it had been placed in the pocket. The radiograph demonstrates the use of the radiopaque material in delineating the marked preponderance of granulation tissue from the soft tissue side of the pocket. Fig. 4C—the material has been removed.

In Figs. 5A, B, C, a series of suprabony pockets was x-rayed in a patient with periodontitis. The radiopaque material outlines the contours of the pocket bases.

In the attempt to remove the material, it has been found that in some instances, remnants may remain behind in some bifurcation and trifurcation involvements. The greater the depth and tortuosity, the more difficult is the removal of the material.

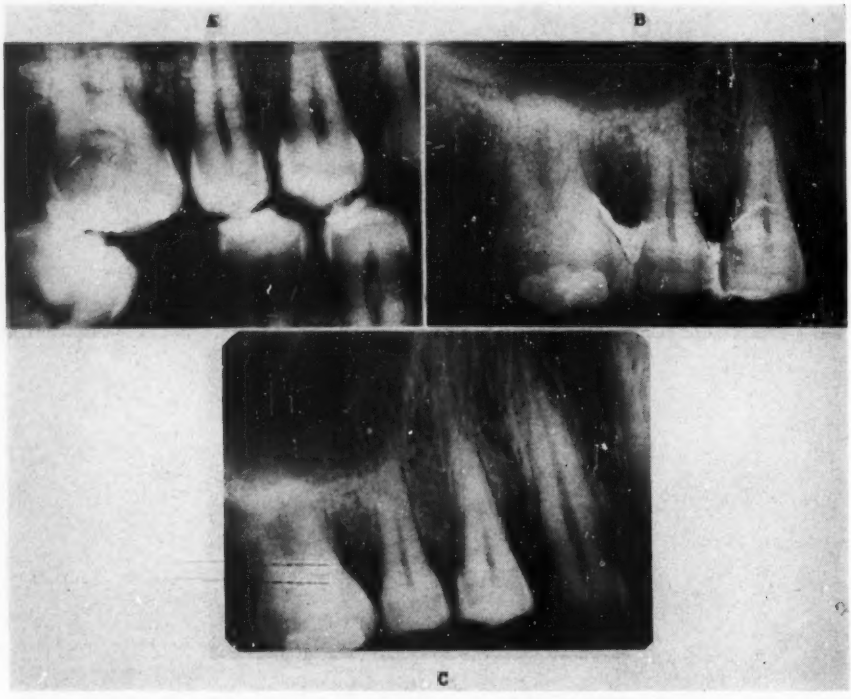

Fig. 5. A. Upper left bicuspid-molar area. B. Same area with radiopaque material in place. Note that material outlines the crevicular bases. C. Same area with material removed.

DISCUSSION

The radiopaque substance described, fulfills most of the criteria required for an effective contrast medium. The rubber base impression material, plus alloy, can be introduced easily into the pocket, and to the full depth of the pocket. The material is non-irritating to the periodontal tissues. It is adequately opaque so that it can be distinguished from the immediately surrounding hard tissues.

An additional desirable feature of this medium is that a three-dimensional replica of the pocket may be obtained before, during, and after treatment has been rendered. This finding opens the way to new possibilities of studying periodontal pockets.

In most instances, the material is readily removable. However, in deep bifurcation and trifurcation involvements, and extensively tortuous pockets, remnants may remain behind. The authors recommend therefore, that the radiopaque material be used primarily as a research tool, rather than a clinical aid.

SUMMARY

An initial report on the development of a radiopaque rubber base-alloy contrast medium has been presented. The technique for proper application of the material has been described. Pockets of varying topographies have been outlined with the radiopaque material and radiographed. A three dimensional replica of a pocket was demonstrated.

The authors wish to express their appreciation to Dr. Henry M. Goldman, who in 1955, suggested the need for an adequate radiopaque material for

use in periodontal pockets, to Dr. Bernard H. Wasserman for suggesting rubber base material, to Drs. Frank E. Beube, Robert Gottsegen, and Leon Eisenbud for their helpful advice in the preparation of this article.

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TEMPLE UNIVERSITY SCHOOL OF DENTISTRY

Temple University School of Dentistry will present the following postgraduate courses for the 1959-60 session:

Clinical Oral Pathology, Dr. Max Jacobs, January 14-16, 1960.

Periodontal Prosthesis, Drs. Morton Amsterdam and D. Walter Cohen, February 8-12, 1960.

Present Day Periodontal Practice, Drs. Jacoby T. Rothner and Neal W. Chilton. One day a week for ten weeks, starting Tuesday, February 16 through Tuesday, April 26, 1960.

Electronic Electrosurgery, Dr. Maurice J. Oringer, March 18-19, 1960.

Advanced Surgical Techniques in Periodontal Therapy, Drs. Saul Schluger and Jack Alloy, April 4-9, 1960.

For application and additional information write to Dr. Louis Herman, Director of Postgraduate Studies, Temple University School of Dentistry, 3223 North Broad Street, Philadelphia 40, Pennsylvania.

MEDICAL COLLEGE OF VIRGINIA

The Medical College of Virginia, School of Dentistry presents a postgradudate course "Clinical Occlusion".

This course, five-day course (February 12-16, 1960) under the direction of Dr. Arne G. Lauritzen, will be directed toward correlating basic principles with clinical procedures. The clinical portion will consist of demonstration of the complete technical and clinical procedures in occlusal harmonization. Class limited to 16. Fee $150.00.

For further information write to: H. T. Knighton, D.D.S., Director of Postgraduate Instruction, Medical College of Virginia, School of Dentistry, Richmond, Virginia.

Effect of Calf Bone on Rate of Bone Regeneration in Tibia of Guinea Pigs

by RICHARD STALLARD, D.D.S. AND WILLIAM RANDALL, D.D.S., *Minneapolis, Minnesota*

THE regenerative powers of a great many substances have been tried on bone and reported in current literature.[1,2,3,4] Tucker[3,4] has shown that bone can be preserved for long periods of time in plasma with a resultant decrease in its foreign body reaction but retaining a certain amount of its osteogenic activity. He has proposed the idea that: bone due to its low metabolic rate will stay alive indefinitely if stored in a normal nutrient medium and refrigerated at 40°F., further that repair actually begins during storage, and finally that bone loses its species characteristics. Major blood vessels have also been successfully despeciated and transplanted in dogs.

The purpose of this study was to ascertain what influence the drug Bo 175* might have on the rate of bone regeneration in the tibias of guinea pigs. It was planned to utilize both hind legs, one as a control, the other treated; the results were determined from serial histological preparations.

Fig. 1. Experimental animal at 12 days. Osteoblasts surrounding bone chip from Bo. 175.

EXPERIMENTAL PROCEDURES

Thirty young guinea pigs were used for this experiment. The animals were anesthetized with Pentobarbital sodium in a concentration of 60 mg/cc. Twenty milligrams per kilogram of body weight was administered by intraperitoneal injection.

A longitudinal incision was made on the medial side of each leg allowing direct access to the tibia. The periosteum was stripped away with a periosteal elevator. Two holes were drilled in each tibia with a number of 560 dental drill. The first hole was placed approximately one-half an inch from the head of the tibia with the second hole one-quarter inch caudal to the first hole. The holes penetrated the marrow cavity. The soft tissue and bony wounds were carefully debrided of blood and debris.

The left leg was chosen arbitrally as the experimental leg while the right served as the control. Both defects in the left leg were filled with the drug, Bo 175. The area was again cleaned and observed to make certain the drug was not dislodged by hemorrhage from the marrow cavity. The incision was sutured. The holes drilled in the right leg were cleared of debris and the incision closed with sutures.

The animals were placed on a normal diet and sacrificed at various stages. The stages were 2 days, 12 days, 19 days, and

*Squibb Cultured Calf Bone for Surgical and Dental Use. This material has been marketed by National Bone and Tissue Laboratories, Inc., Houston, Texas, under the name "Osteogen." Material donated by the Medical Division of E. R. Squibb & Sons, New Brunswick, New Jersey.

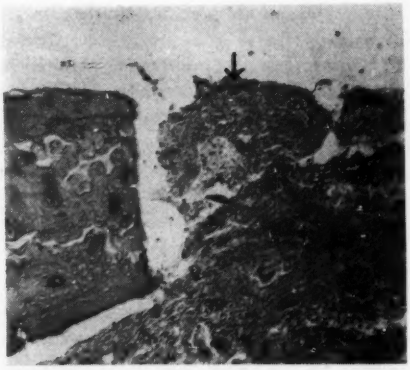

Fig. 2. Control animal at 12 days, normal clot is present.

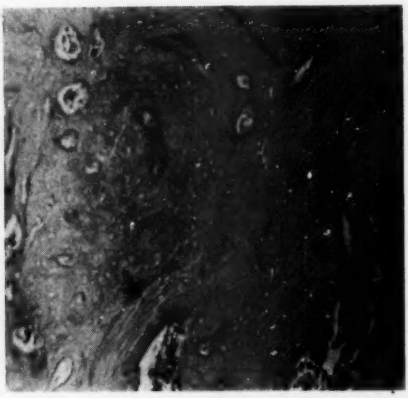

Fig. 3. Experimental animal at 6 weeks. Bone chips are seen as islands of mature bone in the new bone.

at 6 weeks. Six animals died of post-operative complications. The tibias were removed and fixed in Lavdowsky solution. Decalcification was accomplished with 5% nitric acid. The tissues were imbedded in paraffin, sectioned and stained with H + E, P.A.S. and Massons trichrome.

RESULTS

Histological examination on the second day following surgery clearly showed no additional inflammation present in the treated tibia beyond that due to the surgical trauma. At twelve days both wounds were interspersed with connective tissue fibers; but the wounds treated with Bo 175 exhibited a greater degree of activity (Figs. 1 and 2). The wound implanted with bone paste already showed osteoblastic activity around the bony particles of the paste (Fig. 1).

The histological condition on the 19th day after surgery demonstrated accelerated rate of regeneration in the treated tibia. The bony particles of the paste now appear to be acting as nuclei for ossification. A layer of new bone with an increased number of osteoblasts has been layed down surrounding the bony particles of the paste. A normal regenerative reaction is taking place in the untreated tibia.

At six weeks nearly complete healing is evident in both tibial wounds (Figs. 3 and 4). On comparison the new bony tissue growth in the untreated wound is more easily discernible than it is in the experimental. In both cases the new bone is less dense. The regenerated bone in the area

Fig. 4. Control at 6 weeks. Healing is complete but very immature.

of the implant is thicker than it is in the untreated tibial wound.

CONCLUSIONS

The results indicate that the rate of bone regeneration may be increased slightly when the bony wound is subjected to a Cultured Calf Bone implant (Bo 175). Deviation from the expected healing rate was not observed in the microscopic examination of the animals sacrificed at 48 hours. Beginning with the group sacrificed at 12 days, healing in the experimental bony wound began to show an increased regenerative activity.

The first histological section which demonstrated considerable increased osteogenic activity appeared at 19 days. Variation in the number of osteoblasts in the experimental wound as compared to the control bony wound clearly indicated that healing was progressing faster in the area surrounding the bone paste.

Microscopic examination of both bony defects at a period of six weeks postoperative showed that healing was nearly complete. The consistent stimulation of osteogenic activity exhibited in the treated bony wound can be attributed to the presence of Cultured Calf Bone.

SUMMARY

This study was undertaken to observe the rate of bone regeneration during normal healing as compared to the rate of regeneration in the presence of a bone paste (Bo 175). Thirty guinea pigs were treated but postoperative complications resulted in the death of six of the animals. The guinea pigs were sacrificed at various experimental stages and histological slides were prepared from the bony areas involved. The experiment was run under relatively well controlled conditions with the comparison of the rate of regeneration made within each animal and within each group sacrificed.

Examination of the histological sections revealed that bony wounds under the influence of Cultured Calf Bone exhibited an initial increase in healing rate but no appreciable effect on total time required for healing. Another factor which become evident was that the bone paste caused no increased inflammatory reaction in the surgical wound, nor was the paste sloughed as a foreign body.

This study was supported by a United States Public Health Service Grant. FD204

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A.A.P. ANNUAL MEETING OUTSTANDING

The 45th annual meeting of the American Academy of Periodontology which was held in the Barbizon Plaza Hotel in New York City September 10, 11 and 12, 1959, was by all measures outstanding. Dr. James P. Hollars was installed as President and Dr. B. O. A. Thomas elected President-elect, and the efficient and faithful Clarke Chamberlain and James E. Aiguier were reelected Secretary and Treasurer. Some details of the meeting will be published in the next issue of the Journal.

INDEX

The Journal of Periodontology, Volume 30, 1959

JANUARY

Alveolar Bone Loss as Related to Oral Hygiene and Age	7	Olav Schei, Jens Waerhaug, Arne Lovdal Arnulf Arno
Bone Graft in the Treatment of Intrabony Periodontal Pocket in Dogs (A Histological Investigation)	17	Isra Yuktanandana
Morphology of Submarginal Calculus	27	Frank G. Everett
Biochemical and Metabolic Determinants of Experimental Periodontal Disease	32	Grodon R. Potter Philip Person
The Interproximal Stimulator—An Adjunct in Periodontal Therapy	44	Sidney Sorrin Jack L. Thaller
The Role of the Dental Hygienist in Periodontal Practice	47	Richard Chace
Indices for Prevalence and Incidence of Periodontal Disease	51	Sigurd P. Ramfjord
The Periodontal Care Concept—Dentistry's New Horizon	60	Irving Charles Stone
Triamcinolone Acetonide in the Treatment of Recurrent Ulcerative Stomatitis	63	Edward V. Zegarelli Austin H. Kutscher Herbert F. Silvers
Instrumentation in Ultrasonic Periodontal Therapy	67	Sol J. Ewen Paul J. Tascher
A. A. P. President's Address, 1958	72	Donald A. Kerr
Committee Reports	74	
Book Reviews	31, 43, 50	
Announcements	26, 59, 66, 71, 83	
Letters to the Editor	86	

APRIL

Periodontal Reaction to Functional Occlusal Stress	95	Sigurd P. Ramfjord Charles A. Kohler
Periodontosis—Associated with Gingival Lesions—in a Child	112	R. A. Cawson
Dr. Coolidge Honored	115	
The Effect of Physiologic and Pathologic Processes Upon Certain Histochemically Detectable Substances in the Gingiva	116	Samuel Turesky Irving Glickman Beverly Fisher
The Effects on the Epithelial Attachment of Extraction of Adjacent Teeth in Macacus Rhesus Monkeys	124	H. E. Simpson
The Surface of Cementum	126	Lambert A. Benson
Epidemiology and Indexing of Periodontal Disease	133	John C. Greene
A Suggested Classification of Common Periodontal Disease	140	Fermin A. Carranza Fermin A. Carranza, Jr.
Lysozyme Content of Human Gingiva and Various Rat Tissues	148	George W. Burnett Susan Gougé A. E. Toye
Book Review	151	
Presence of Acetylcholinesterase in Human Gingiva	152	James K. Avery Robert Rapp

RK1
J855

Bruxism Splint. Technique Using Quick Cure Acrylic	156	Harold E. Grupe
Effect of Somatotropic Hormone on Gingival Wounds in Normal and Protein Deprived Rats	158	John J. Gromek S. S. Stahl
Book Notice	162	
A Permanent Fixed Amalgam Splint	163	Ralph S. Lloyd Paul N. Baer
Arthur F. Lufkin	165	
Announcements	132, 139, 147, 155, 166, 168	

JULY

Arthur H. Merritt	174	
Repair of Dento-Gingival Junction Following Surgical Intervention	180	Nicholas R. Marfino Balint J. Orban Frank M. Wentz Sol Haberman
Inflammatory and Non-inflammatory Responses to Gingival Invasion by Microorganisms	190	
Effects of Certain Factors on Bacteremias Resulting from Gingival Resection	196	Benjamin Vargas C. Kenneth Collings Lucy Polter Sol Haberman
Degree of Keratinization and Glycogen Content in the Uninflamed and Inflamed Gingiva and Alveolar Mucosa	208	M. D. Weiss J. P. Weinmann
A Study of the Gingival and Periodontal Conditions of a Group of People in Chieng Mai Province	219	J. Meyer M. R. Nibhasara Ladavalya
Case Report—Leukemia Involving the Oral Cavity	223	Robert Harris Irwin Walter Scopp
Papillon-Lefevre Syndrome: Precocious Periodontosis with Associated Epidermal Lesions	230	Arthur M. Quart John I. Ingle
Salivary pH Examination Amongst Indian Children and Its Relation to the Incidence of Periodontal Disease	238	M. K. Sanjana F. S. Mehta R. H. Doctor B. C. Shroff
Epidemiology of Diabetes Mellitus: I. Review of the Dental Literature	242	R. C. Sheridan, Jr. E. Cheraskin F. H. Flynn
Influence of Injury and Adaptation of the Periodontal Ligament to Pathologic Changes During Experimental Lathyrism	253	A. C. Hutto Alvin F. Gardner
Editorial	265	
Announcements—Program for Annual Meeting American Academy of Periodontology	266	
Letters to the Editor	272	

OCTOBER

See page 276 of this issue.

16941